Changes to Associative Learning Processes in Later Life

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Abstract

The present research sought to describe and explain age related changes to associative learning processes. Eleven experiments were conducted using a human conditional learning paradigm. Background data on health, lifestyle, and cognitive ability were collected and used as predictor variables in multiple regression analyses.

Experiments 1 to 8 were formative, and found that older participants showed an overall age related decline in learning ability exacerbated by the number of stimuli and outcomes used, and the concurrent presentation of different problem types. Configural models of learning (e.g. Pearce, 1994, 2002) best predicted young participants' learning whereas older people's learning was more consistent with elemental models (e.g. Rescorla-Wagner, 1972), suggesting an age related change in generalisation processes. Those who learned problems better were also more likely to be able to articulate a rule that had helped them learn the problem. Age itself was the most predominant predictor of accuracy in these experiments.

Experiments 9, 10, and 11 were multiple stage experiments that looked at the extent of pro- and retro-active interference in learning. Experiments 9 and 10 used easy and hard HCL problems to examine the role of rule induction in learning. Older participants who had learned initial discriminations better were more prone to pro-active interference in both experiments, the extent of which was predicted most reliably by fluid intelligence. Rule learning had a profound effect on participants' predictions during the unreinforced test stage. In Experiment 9 (Easy-Hard) younger participants suffered from more retroactive interference than older people. This pattern was far less pronounced in

Experiment 10, (Hard-Easy) suggesting that problem order affected the way participants generalised from rule-based knowledge. This observation is inexplicable by associative learning theories, and explanation may require a problem solving approach. Experiment 11 examined feature-based generalisation. Again older participants suffered more proactive and retroactive interference and elemental theories predicted their responses best, whereas younger participants responses were consistent with configural models of learning. In this instance, resistance to pro- and retro-active interference was predicted by fluid intelligence.

Overall the research concluded that there is a demonstrable, complexity dependent change in associative learning processes in later life. It appears that humans have an increasing tendency to rely on elemental, rather than configural processes of generalisation in later life, and this leads to overgeneralisation between stimuli and an inability to resist pro- and retroactive interference in learning. This may be as a result of an inhibitory or source monitoring failure as a consequence of atrophy in the frontal lobes of the brain, although some of the learning deficits are explicable through mnemonic decline.

Chapter 1: Introduction to Associative Learning

1.1 Fundamentals

Associative learning in its broadest sense has a long history. Aristotle (c.350 BC) suggested that ideas experienced together tend to be remembered together through the principles of contiguity, similarity, and contrast. In this conception associative learning is simply the strengthening of mnemonic connections between subjective experiences that happen to be either very similar, very different, or close together in time and space. Later Ebbinghaus (1913) tested Aristotle's conjectures through introspection, and agreed that these fundamental principles held under more rigorous inspection. Obviously, though, both men relied on subjective methodologies and this can be seen as a fundamental flaw in their investigations on the subject.

Associative learning in the context of this thesis, however, involves creating an association between a stimulus and a response such that when the same stimulus is encountered again it will evoke the same response. Ivan Pavlov (1927; see also Mackintosh, 2003, and Wasserman & Miller, 1997 for reviews) was a Russian physiologist who researched the digestive systems of dogs and pioneered the study of classical conditioning, a variant of associative learning. Crucially, he devised an objective experimental approach to investigate the formation of this kind of association. In his experiments a bell was rung each time the dogs were brought food. The bell on its own had not provoked any response prior to the experiment, but subsequently was enough to elicit salivation by itself. The dogs had learned to associate the sound of a bell with food, and the sound of a bell was enough to trigger the normal physiological reaction to the presence of food. Classical conditioning could therefore be said to be the learning of associations between stimuli and physiological reflex responses. Pavlov introduced new terminology to describe the different components of classical conditioning. Food was the *unconditioned stimulus* (US), which needs no conditioning to evoke the dogs' salivation, or *unconditioned response* (UR). Initially the bell was a *neutral stimulus* (NS), but after conditioning becomes the *conditioned stimulus* (CS), which may evoke salivation on its own: the *conditioned response* (CR). He also noted that subsequent repeated presentation of the CS in the absence of the US led to the animal's response to the bell alone disappearing: a process he called *extinction*. Furthermore it was also recorded that the animals made similar responses to similar stimuli, a phenomenon known as *generalisation*.

Most theorists (e.g. Pavlov, 1927; Estes, 1959; Rescorla & Wagner, 1972; Rescorla, 1979; Pearce, 1987; Wagner, 2003; LePelley, 2004) argue that the conditioning process leads to the establishment of a connection, or association, between internal representations of the CS and US. Furthermore the magnitude of this association increases with repeated paired presentations (Dickinson, 1980), and is linearly reflected by the accuracy of CRs. One way of investigating classical conditioning is to present participants with two CSs, one of which, stimulus A, is followed by the US (A+), whilst the other, stimulus B, is not (B-; note that 'A' and 'B' and '+' and '-' are abstract labels that could signify any stimulus or outcome type and that this kind of notation is commonly used in the associative learning literature). Under these circumstances one would expect a CR when A+ is presented, but not when B- is presented. This simple paradigm is known as discrimination learning, and clearly one would surmise that an association had been formed between stimulus A+ and the US, but not between B- and the US. Although much has been learned from discrimination learning experiments this simple task is unable to test the relative accuracy of the predictions of an abundance of theories seeking to explain fundamental learning principles. There are several more complex paradigms currently in use, for instance negative (NPP) and positive (PPP) patterning problems (see e.g. Jarrard, 1993; Kehoe, 1988; Shanks, Charles, Darby, & Azmi, 1998); and biconditional problems (see e.g. Saavedra, 1975; Shanks, Charles, Darby, & Azmi, 1998). All these experimental designs involve compound training, where two stimuli are presented together to form a unique compound CS. NPPs, for example, require participants to learn that a stimulus presented alone, or as an element, predicts the US (e.g. A+, B+), but when presented as a compound (e.g. AB-) they do not. Conversely in PPPs compound stimuli (e.g. CD+) predict the US, but their elements (e.g. C-, D-) do not. Biconditional problems, on the other hand, consist entirely of compound stimuli arranged such that each element in the problem is associated with the presence and absence of the US equally (e.g. AB+, CD+, AC-, BD-). One important observation concerning both these classes of problem is that they require non-linear solutions: one cannot reliably predict associations between the US and compound stimuli by summing the associative strengths of elemental stimuli. As we shall see this property makes it possible for these problems and others like them to distinguish between extant theories of associative learning.

There are two broad classes of theory that seek to explain associative learning. The first class has been referred to as ‘nonselective elemental’ theories, and the second as ‘configural theories’ (Rudy & Sutherland, 1989). The former are elemental in that they assume that summing the associative strength of their constituent elements, or components such as brightness, size, modality et cetera, predicts responses to stimuli. They are nonselective in that all elements or components within the stimulus have an equal chance of association with the US. For the remainder of this thesis these theories (e.g. Spence, 1936; Estes, 1950; Rescorla & Wagner, 1972; Mackintosh, 1975; Wagner, 2003; LePelley, 2004) will simply be referred to as elemental theories. Configural theories on the other hand take a Gestalt approach and assume that each stimulus configuration constitutes a separate exemplar: even if two elements are combined to form a compound these are treated as separate stimuli (e.g. Pearce, 1987, 1994, 2002; Kruschke, 1992). Yet other theories attempt to bridge the gap between elemental and configural accounts by integrating aspects of both classes of model with neuropsychological accounts (e.g. Gluck & Myers, 1993; Sutherland & Rudy, 1989; Rudy & Sutherland, 1995).

The remainder of this chapter will be devoted to an appraisal of elemental theories, and Chapter 2 will examine configural theories, assess the relative success of each class of theory in explaining fundamental learning processes, and look at neuropsychological models of learning. Chapters 3 and 4 go on to explore the effects of ageing on cognitive ability and to speculate on the likely effects of the ageing process on people's capacity for associative learning.

1.2 Elemental Theories

As stated before elemental theories make the assumption that each element present on a trial is processed separately and acquires its own associative strength. Furthermore the separate associative values of all elements presented together as a compound can be summed to predict a response: a principle known as ‘summation’. These assumptions were clearly made by researchers prior to the development of computational models of learning. Spence (1936), for instance, proposed that the total excitatory strength of a stimulus compound was a function of the sum of the associative strengths of its elements. This obviously reflected both of the assumptions inherent in elemental models; that firstly the elements in compound stimuli are processed separately, and secondly that by summation the associative strengths of compound elements can be added together to predict an organism’s response.

Spence (1936, 1937) also developed the idea that inhibition contributed to associative learning. As above he predicted that rewarding an animal for responding to stimuli S+ would lead to an increased likelihood of responses on subsequent presentations of the same stimuli. In addition he also proposed that responses to unreinforced stimuli S- would be incrementally inhibited in the same way, leading to an increasingly decreased likelihood of responses to that stimulus. Spence (1937) also generated ideas about the nature of generalisation: or the tendency to respond in similar ways to perceptually similar, but not identical, stimuli. In this paper he reports the finding that animals could discriminate between two light stimuli (S+; S-) varying on a single dimension of brightness. Spence (1937) proposed that each stimulus would generate its own generalisation gradient: a curve approximating the normal distribution that predicted the strength of the animal’s responses. Furthermore he suggested that the excitatory generalisation gradient corresponding to the stimulus S+ would be larger than the inhibitory generalisation gradient surrounding the stimulus S- and animals would therefore be more likely to respond to S+ than S-. Additionally Spence’s theories made the prediction that any subsequent stimuli S’ with an intensity displaced from S+ in a direction away from S- will result in a stronger response than to S+ alone, since the loss of associative strength incurred by a move away from S+ is less than the reduction in inhibition resulting from the move away from S-. This 'peak shift' phenomenon has been confirmed by a number of studies (e.g. Honig, 1962; Sachs, 1969). One much cited example of empirical support for both generalisation and the peak shift phenomenon is Hanson (1959). In this study pigeons were trained to discriminate between lights of various wavelengths: responding to lights of 550nm (S+) but not to lights of 560nm (S-). When tested without reinforcement the pigeons responded more strongly to lights of 530nm and 540nm than to lights of 550nm, although the original discrimination was preserved and the pigeons responded least to lights of 560nm and greater. The concepts of generalisation and inhibition are fundamental to this thesis, and will be expanded on in Chapter Two in the context of configural models, and in Chapter 4 in relation to cognitive ageing.

Spence's contribution was seminal and parsimonious. His theories made specific, testable predictions although there are some obvious drawbacks. His view of inhibition was flawed since he assumed that this is merely the result of non-reinforcement, whereas inhibition is clearly a more complex phenomenon that this analysis implies (see e.g. LoLordo & Fairless, 1985). Furthermore the theory fails to predict responses for even the simplest non-linear problems, such as the feature positive problem AB+, B-. According to Spence's theory this discrimination should be insoluble since it is assumed that both elements enter into an association with the US, and both elements A and B would be completely, or asymptotically, associated with the US. Consequently an animal would make a CR to the B- element when presented alone. A raft of empirical evidence exists to contradict this prediction: animals *can* learn an AB+, B- feature positive discrimination (e.g. Wagner, 1969; Reberg & Leclerc, 1977). Another more fundamental weakness of Spence's theory is the assumption that generalisation occurs only between stimulus elements that vary along a single dimension, such as brightness or tone.

A slightly different approach that perhaps takes a more realistic view of both generalisation and the effects of non-reinforcement was that of Stimulus Sampling Theory (SST: Estes, 1950; Atkinson & Estes, 1963). SST proposes that the background context of a CS was encoded along with the CS itself, and later that the only effect of non-reinforcement was to weaken previously established associations. The theory suggests that when stimuli A+, B- are presented the context of that presentation, X, also acquires associative value, and an organism responds to stimuli on the basis of the statistical probability of it predicting the US. A simple discrimination now becomes an AX+, BX- problem, and one consequence of this is that the theory predicts that either discriminations shouldn't be learned perfectly, or that context X should ultimately lose its associative value, since B can do nothing other than lose acquired associative value through non-reinforcement. The lack of clarity surrounding any mechanism through which simple discriminations can be acquired is therefore a definite downfall to this theory. Furthermore SST suffered from the same basic problems as Spence's theory in that it is unable to predict solution of the feature positive problem. Given an AB+, B- discrimination the model predicts that since element B has an equal probability of being present on both reinforced and unreinforced trials it will be 'sampled' as having a positive valence on some B- trials and a negative valence on some AB+ trials, rendering the discrimination impossible to acquire. Despite this prediction the empirical evidence suggests that animals can learn feature positive problems (Reberg & Leclerc, 1977), as well as the more difficult negative patterning (Jarrard, 1993) and biconditional problems (Saavedra, 1975).

One of the fundamental weaknesses of both Spence's theory and the SST approach was that these theories predict that elements of a compound AB+ would both acquire an equally powerful, equally complete association with the US. Another, more technical way to put this would be that an organism's learning would reach asymptote at an associative strength, λ, of 1 (usually expressed as 1λ) for both elements of a compound, each of which alone would therefore always predict an outcome. This leads to an inability to predict the solution of a feature positive problem, as well as other problems such as blocking (Kamin, 1969): a paradigm where an experimental condition consisting of Stage 1 A+, AB- trials leads to reduced US predictions to stimulus B at test relative to a control condition of Stage 1 AB- trials. Clearly this is another situation where neither Spence's theory nor SST could predict the outcome. Both feature positive problems and blocking are predictable, however, if one assumes a competitive learning rule. According to models that assume cue competition the elements of a compound compete with each other for an association of finite magnitude, usually assumed to be 1λ. Given a feature positive problem AB+, B- stimulus element A would acquire most of the associative strength, allowing a model to predict B-. Similarly when applied to blocking a competitive rule allows stimulus element A to once more gain the entire associative valence and allow the model to predict retarded responding to element B at test. The fundamental assumptions of cue competition were outlined by Konorski (1948, 1967) and have spawned many models of learning since (e.g. Rescorla & Wagner, 1972; Mackintosh, 1975; Pearce & Hall, 1980; Wagner, 2003; LePelley, 2004). These models share with Spence's theories and SST the assumptions of summation and of elemental processing, although all assume the key difference of cue competition.

Without question the most influential model of learning to have emerged over the last fifty years is the Rescorla-Wagner (1972) model. This model is fundamentally based on Widrow & Hoff's (1960) Delta rule, developed to minimise errors in radio signals. The Rescorla Wagner (1972) theory uses the assumption that the learning process is essentially an algorithm that minimises the difference between what is predicted to happen, and what actually happens. The theory incorporates several of Spence's assumptions: that learning is an incremental process leading to an increased likelihood of responding to a reinforced stimulus involving the analysis of stimulus elements that can predict responses to compound stimuli through summation. Furthermore the theory assumes a finite strength of an association between the CS and US, which is said to be at asymptote when at its maximum strength, usually represented as the Greek letter λ. As stated before the key difference is that Spence assumed each element of a compound stimulus could reach asymptote, whereas the Rescorla-Wagner model assumes that each element in a compound competes for a finite level of association determined by the presence of the US. A consequence of this is that the sum of a compound's elements can equal no more than λ, usually assumed to be 1. Another feature of the theory is that novel, or surprising stimuli result in a greater increase in associative strength than unsurprising or previously seen stimuli, allowing the theory to predict a classic, negatively accelerating 'learning curve'.

Fundamentally the Rescorla-Wagner theory is an error correction algorithm implemented in a two layer neural network. The equation representing the change in associative strength for a single element between the CS and US, ΔV_n for each trial is shown below in Equation 1.1.

Equation 1.1 $$\Delta V_n = \alpha\beta(\lambda - V_n)$$

The Rescorla-Wagner theory therefore predicts that the change in associative strength on any trial is equal to the ceiling of associability for the US λ (λ= 1 if the US is present, λ = 0 if the US is absent) minus the existing associative strength, thereby allowing novel stimuli to acquire greater increases in associative value than existing stimuli. The model also incorporates two further parameters α and β, which denote the conditionability of the CS and US respectively. These parameters represent the learning rate of the model. Generally speaking β is set at 1 whereas α is set at around 0.1 to prevent the model merely predicting a 'stepwise' alternation of the strength of association between 0 and 1.

Things become slightly more complex when dealing with compounds. Here, as stated before, the model assumes that elements in a compound stimulus cannot accrue more associative strength than the ceiling of associability λ. Hence the increase in associative strength between the CS and US for each element in a compound AB+ is determined by Equations 1.2 and 1.3, below.

Equation 1.2 $$\Delta V_A = \alpha_A\beta(\lambda - V_{AB})$$

Equation 1.3 $$\Delta V_B = \alpha_B \beta(\lambda - V_{AB})$$

Consequently the change in associative strength between CS and US for compound AB+ can be expressed as in Equation 1.4, below.

Equation 1.4 $$\Delta V_{AB} = \alpha_A \beta(\lambda - V_{AB}) + \alpha_B \beta(\lambda - V_{AB})$$

The Rescorla-Wagner theory correctly predicts the solution of both feature positive and blocking problems. Taking feature positive problems as an example conditioning to the AB+ discrimination would follow the pattern indicated by Equation 1.4. In the normal course of events this would lead each element to have an associative value of 0.5, assuming $\lambda = 1$. Introduction of the B- discrimination, however, would lead to stimulus B acquiring no associative strength since here $\lambda = 0$, as the element is unreinforced. This leaves stimulus A to gain an associative strength equal to λ as the best predictor of the US, since compound AB must have an associative strength equal to λ and is the sum of the associative strengths of elements A and B.

The Recorla-Wagner theory was, however, primarily developed to deal with blocking (Kamin, 1969), a paradigm where an experimental condition consisting of Stage 1 A+, AB- trials led to reduced US predictions to stimulus B at test relative to a control condition of Stage 1 AB- trials. The model's success in predicting responses in blocking paradigms is partly due to the assumption that the response to a compound is predicted by summation of the responses to its elements, and that the ceiling of associability λ forces elements within compound stimuli to compete directly for associative strength. This allows A+ to gain associative strength while B proportionately loses associative strength to allow prediction of AB-. At test, therefore, responses to B will be reduced, or 'blocked'.

The experiments Kamin (1969) conducted on blocking, in common with most other researchers in the field of associative learning, used animals as participants. One important development that has occurred relatively recently is the application of associative models of learning to Human Conditional Learning (HCL). Dickinson, Shanks & Evendon (1984) were the first to demonstrate that cognitive responses to external events or stimuli may be equivalent to the physiological responses seen in 'pure' classical conditioning or the behaviours observed in operant conditioning. Their demonstration that human category judgements are subject to the blocking phenomena has been reliably replicated (e.g. Dickinson & Shanks, 1985; Chapman & Robbins, 1990; Mitchell & Lovibond, 2002), suggesting that associative processes may underlie causal judgements in humans. In the last twenty years HCL has been subject to much investigation (see e.g. De Houwer & Beckers, 2002; Dickinson, 1994, 2001 for reviews), and the parallels between HCL and animal conditioning experiments continue to receive experimental investigation. For instance Shanks, Charles, Darby & Azmi (1998) used a 'food allergy' paradigm to investigate HCL. Here participants are given repeated exposure to foods both on their own (*elements*) or in pairs (*compounds*) and are asked to predict whether the food or foods will cause an allergic reaction or not. Following each prediction participants are given feedback telling them whether the food or foods caused an allergy, and their task is to learn the contingencies between foods and allergies. For example Eggs or Milk alone (i.e. as an element) may lead to an allergy, but when presented together as a compound (i.e. Eggs *and* Milk together) they may not: forming a Negative Patterning Problem and yielding similar results to animal studies of classical conditioning (e.g. Jarrard, 1993). The findings of these experiments will be discussed in more detail later on, but since this paradigm forms the focus of the investigation contained in this thesis it is important to describe the broad methodology and the comparisons between HCL and classical conditioning early on.

Despite the obvious improvements in terms of prediction the Rescorla-Wagner model made over theories such as Spence's and SST there are still problems with it, particularly in terms of its elemental assumptions. While elemental assumptions may be considered a strength as far as blocking is concerned; they can be a drawback in terms of non-linear problems. Any situation where the elemental assumptions of the model are violated will result in inaccurate predictions. For instance in Positive Patterning Problems (PPP) elements predict no outcome, whereas their compounds do (e.g. A-, B-, AB+). The standard Rescorla-Wagner model cannot correctly predict responses here; since all elements are present on both reinforced and non-reinforced trials they each gain an associative value of 0.5. Assuming that an activation threshold of the CR must be greater than 0.5 for the model to predict the presence of a US, however, allows solution of a PPP. In this case neither element alone would suffice to predict a CR, but the sum of the elements would equal λ, and thereby predict the arrival of a US. This solution, however, would not hold for a Negative Patterning Problem (NPP), where elements predict the US, but compounds do not (e.g. A+, B+, AB-). Despite this there are extant empirical data demonstrating the ability of humans (e.g. Shanks, Charles, Darby, and Azmi, 1998), and animals (e.g. Rudy & Sutherland, 1995; Jarrard, 1993) to solve these discriminations (even concurrent NPP and PPP in the case of Shanks et al.). Equally problematic are biconditional discriminations where each element is associated with the US (+) or No US (-), depending on configuration of elements (e.g. AB+, CD+, AC-, BD-). Clearly no rule of summation or response threshold can predict responses here, yet empirical data suggest both humans (Shanks et al. 1998) and animals (e.g. Rudy et al. 1995, Saavedra, 1975) can learn these discriminations.

One way of dealing with these data while retaining the parsimony of an elemental approach is the unique cue theory (Rescorla, 1973). Here compounds are assumed to acquire an extra element, or 'unique cue' when amalgamated. A unique cue can acquire its own valence to allow for the kind of anomalies inherent in non-linear problems. In a negative patterning problem the elements have a positive valence (i.e. A+, B+) while the compound has negative, or no valence (AB-). By adding the negatively valued unique cue X to the compound AB, making ABX, the positive valence of its elements can be counteracted, rendering non-linear problems such as negative and positive patterning problems soluble. There are some problems, however, with unique cue theory, since firstly there is no specification of how, when, or why unique cues would form. Moreover under certain conditions unique cue theory erroneously predicts retroactive interference in learning. This is where subsequent learning effects original learning. Unique cue theories predict, for instance, that learning Stage 2 B+ discriminations following Stage 1 A+, AB- trials should result in increased responding to the AB compound at test, albeit to a lesser extent than 'simple' elemental theories. Shanks, Charles, Darby, and Azmi (1998) found little evidence to support these predictions in an undergraduate population using a food allergy task. Participants were asked to remember which food or pair of foods led to an allergy, and which to no allergy. Shanks, Charles, Darby, and Azmi (1998) conducted further experiments specifically designed to induce elemental processing and found no evidence that elemental processing interfered with subsequent configural learning. They then gave four groups of participants different pre-treatments designed to induce different styles of processing, or cognitive set, before giving them an interference experiment. One group received a biconditional pre-treatment (WX+, XY-, YZ+, WZ-), another a conditional pre-treatment (WX+, WY+, XZ-, YZ-), another group a double element discrimination (W+, WX-, X-, Y-), and lastly a further 'explicit' group a linearly soluble discrimination (W+, WX+, X-, Y-). Clearly the 'explicit' pre-treatment group was designed to promote elemental processing, and was expected to influence later processing. Following this all groups received Stage 1 A+, AB-, AC+ discriminations and subsequently a Stage 2 discrimination of the form B+, DE-. Afterwards, participants received ten test trials of the form A+, AB-, AC+. Despite an admirable attempt to induce interference of the sort predicted by elemental models of learning Shanks, Charles, Darby, and Azmi (1998) failed to find any differences between the pre-treatment groups at test: all groups had preserved the Stage 1 discrimination.

A more recent variation on the elemental theme was proposed by LePelley (2004) on the assumption that a cue's associative history dictates the extent to which it is attended to as well as the saliency of that cue. In this conception attention is viewed as the weight which is given to a particular stimulus relative to other potential stimuli and determines which stimuli have access to the learning process and which do not. This part of the model is based on an extended version of the Mackintosh (1975) model developed by LePelley (2004) to account for empirical phenomena such as learned irrelevance, where stimuli that are good predictors acquire associative strength at the expense of stimuli that are not, whilst preserving its ability to predict other empirical phenomena such as blocking. Sometimes, however, learning is faster when stimuli are poor predictors of external events (e.g. Wilson, Boumphrey & Pearce, 1992) so the model proposes, after Pearce and Hall (1980), that saliency dictates the rate at which stimuli should be learned about.

This model represents an improvement over the Rescorla-Wagner (1972) version in terms of its ability to predict a wider range of empirical phenomena and takes into account the way conditional as well as unconditional stimuli are processed. It does, however, contain more parameters that need to be dictated by the experimenter and is therefore more arbitrary. Another point is that, despite its relative complexity, the model remains fundamentally elemental in its assumptions. This means that the model cannot predict empirical observations such as the solution of biconditional problems by animals (Saavedra, 1975) and humans, or the resistance to retroactive interference found in human participants (Shanks, Charles, Darby, and Azmi, 1998).

It is clear from this brief review that elemental theories describe some learning phenomena well. Blocking paradigms demonstrate that people and animals can make elemental assumptions. Non-linear paradigms insoluble by elemental means, however, show that when these elemental assumptions are contradicted young people and animals have little difficulty in learning a solution that allows them to predict the presence of a US, and for this learning to be preserved in the face of concerted experimental effort to disrupt it. Another class of theory has been developed to account for these situations that assume that elements and their compounds are distinct entities. These theories are known as Configural, or exemplar based, theories since they suggest that any configuration of stimuli, or exemplar, is a unique combination that develops its own separate relationship with the US. The next chapter will continue by looking first at exemplar based models, before going on to examine other models of learning and a closer look at the applicability of these models to human contingency learning.

Chapter 2: Configural and Neuropsychological Theories

2.1 Configural Theories

Given the failure of elemental theories to predict observed responses to non-linear problems in animals and humans it is unsurprising that researchers have derived alternative models based on different assumptions. Configural, or exemplar-based, theories (e.g. Pearce, 1987, 1994, 2002; Kruschke, 1992) assume that elements and compounds constitute distinct entities, or exemplars, capable of acquiring associative strength independently. This approach is clearly superior to elemental models when predicting responses to non-linear problems. Consider the biconditional problem AB+, CD+, AD-, BC-. Assuming that each compound accrues associative strength independently, regardless of the associative value of its elements, enables a model based on strict configural assumptions to correctly anticipate the solution to this problem. A major flaw with this assumption is, however, that without some generalisation between similar stimuli, empirical phenomena such as blocking remain inexplicable. Furthermore, it is unrealistic given the well-documented empirical demonstrations of generalisation according to perceptual similarity detailed in the preceding chapter (e.g. Spence, 1937; Hanson, 1959). The correct prediction of blocking requires the generalisation of associative strength between an element A+ and a compound AB-, such that the compound element A acquires all the associative strength. This allows a model to foresee the response of animals or humans to the problem: that later responses to element B are retarded in comparison to groups that only receive AB- discrimination training earlier on. It is clear from this that blocking requires an assumption of almost complete generalisation between similar stimuli. At the same time other empirical phenomena, such as the solution of PPP, NPP, and biconditional problems (e.g.

Shanks, Charles, Darby, and Azmi, 1998) require a suspension of generalisation between similar stimuli. Therein lies a contradiction, and one of the major problems for all learning theories: when, how, and under what circumstances are responses to stimuli generalised between elements or not, and how can a computational model learn to discriminate between these circumstances? Generalisation is a difficult problem for associative learning theories to address, for reasons that should become increasingly apparent.

Theoretical definitions of the concept of generalisation are equivocal, although it is clear that it is an important idea. Some of the empirical bases for generalisation, such as Spence (1937), and Hanson (1959) have already been described. For another example consider Guttman & Kalish (1956), who found that pigeons trained to respond to a yellow light subsequently responded most strongly to yellow light, next most strongly to yellow-green and yellow orange light, and least strongly to green or orange lights. It is not unreasonable to suppose the observation that frequencies of responses increase as stimuli become more similar to the conditioned stimulus can be explained by automatic spreading activity between representations of similar stimuli. For some, therefore, (e.g. Pavlov, 1927; Mackintosh, 1974, 1975, 2003) generalisation means the automatic spread of activation to adjacent cortical areas, and therefore to representations of similar stimuli. This is close to Hebb's (1949) idea of spreading activation through the strengthening of connections, or synapses, between neurons, although Hebb's theory is based on correlation of activity between neurons rather than their physical proximity. Others (e.g. Lashley & Wade, 1946), however, suggest that generalisation occurs as the result of an inability to discriminate between stimuli, rather than an automatic spread of activation. Lashley and Wade (1946) would argue that the pigeons in Guttman et al.'s (1956) experiment responded to similar lights not because of the prediction that a response would be automatically expected because of a superficial similarity between stimuli, but because the stimuli may be difficult to tell apart, or consciously discriminate between. Generalisation in this view is a matter of making the same response to a stimulus that is effectively indistinguishable from an earlier CS. Whichever view one accepts; the practical implications are the same in that generalisation appears contingent on perceptual similarity.

Some configural models, such as Pearce (1987, 1994, 2002) deal with generalisation by suggesting that some of the associative strength of a stimulus is generalised to all other stimuli with non-zero perceptual similarity. Pearce's model assumes that any perceived change to a trained CS will result in a change to the evoked CR of a magnitude equal to the extent of change to the perceptual attributes of that CS. Certainly this view is consonant with Guttman and Kalish's (1956) data described earlier: a change in the wavelength of the CS resulted in a decrement in the CR equal to the difference between stimuli. In addition Mednick & Friedman (1960) demonstrated an equivalent effect using auditory tones: responses to stimuli in both cases seem to be dictated, at least in part, by a superficial similarity between the present stimulus and those seen before. It seems reasonable; therefore, to make the assumption that novel stimuli are responded to in terms of their perceptual similarity to existing stimuli through generalisation. Pearce's (1994) connectionist model assumes that CS input units feed activation forward to internal layer output units, and thence to a configural layer, which enters into associations with the US. Input layer activations have values of 0 and 1 for stimulus presence or absence. Output layer activity o_i is, assuming an input pattern of 0 or 1, calculated using Equation 2.1 below, where η is the number of input units active on each trial.

Equation 2.1 $$O_i = \frac{1}{\sqrt{\eta}}$$

This has the effect of making the activation of output units for each element contingent on the number of elements active at the time. In essence the model gives a single element input a value of 1 at output, but less than 1 if it is part of a compound. For instance each element of the compound AB would give an output of 1 to the configural layer if present alone, but a value of 0.71 if presented as part of a compound. The calculation of the activation of the configural unit representing individual elements or compounds α_j is given in Equation 2.2.

Equation 2.2 $$\alpha_j = \Sigma w_{ij} \cdot o_i$$

Pearce (1994) assumes that the strength of the connection w_{ij} from the output to configural nodes is equal to the output activation of the output unit. Thus if the element A was presented alone the output to node *j* representing the element would equal 1, and the sum of all the weights multiplied by outputs would also equal 1. If compound AB was presented, however, the activation of the configural unit for each element would only equal 0.5, but the activation of the node representing AB would equal 1. The strength of the association between the configural node **A** and the US is dealt with using a modified form of the

Rescorla Wagner (1972) rule (c.f. equations 1.1, and 1.2). Equation 2.3 shows how the change in the weight of the connection between a configural unit A and US $\mathbf{E_A}$ is calculated.

Equation 2.3 $$\Delta E_A = \alpha\beta(\lambda - V_A)$$

The US prediction for configural unit A, $\mathbf{V_A}$, is shown in Equation 2.4, below. Here $\mathbf{E_A}$ is the existing weight of the connection between configural unit A and the US. $\Sigma_A \mathbf{S}_{,i}$ is the sum of the measures of similarity between pattern A and other configural units *i* activated by pattern A (i.e. which share elements with pattern A). Here $\mathbf{E}_i$ is the associative strength of each of the configural units activated by pattern A.

Equation 2.4 $$V_A = E_A + \Sigma_A S_i \cdot E_i$$

Equation 2.5, below, shows the critical calculation of similarity between pattern A and pattern B, $_{A}\mathbf{S}_{B}$. This is calculated as a ratio of the number of elements pattern A and B share, $\boldsymbol{n_C}$, and the product of the number of elements present in pattern A, $\boldsymbol{n_A}$, and the number of elements in pattern B, $\boldsymbol{n_B}$.

Equation 2.5 $$_{A}S_{B} = \frac{n_{c}2}{n_A \cdot n_B}$$

In this way Pearce (1994) suggests that US prediction is partly a function of the direct association between a pattern of activation A in the configural layer of the model and the US, and partly the result of generalisation of associative strength from other perceptually similar stimuli. Although other configural units ***i*** will not change the weights of their connection to the US during presentation of stimulus pattern A the associative value of their existing relationship to the US is considered in Equations 2.4 and 2.5, and the value of $\mathbf{V_A}$ limits the extent of any change of associability between pattern A and the US. Critically, Pearce (1994) gives results of simulations demonstrating the model's ability to predict blocking, where pre-exposure to A+ prior to AB+ contingencies leads to reduced responses to B at test relative to pre-exposure to only AB+ contingencies, feature negative (A+, AB-), and feature positive (AB+, A-) discriminations. Consider the feature positive problem as a simple example. The net associative strength of AB, V_{AB} needs to reach a value of 1λ, and the net associative value of A, V_A should equal 0λ, before learning reaches asymptote and changes to associative strengths cease. To achieve this the model assumes that V_A will reach a value of less than 0λ to counteract the positive associative strength generalised from AB, and V_{AB} a value greater than 1λ to oppose the negative associative strength of A. The similarity of AB to A and of A to AB is 0.5 in both cases so, after accounting for this generalisation, the associative strength of AB would be 1.33λ and the value of A would be –0.66λ since $V_{AB} = 1.33 + (0.5 \cdot -0.66) = 1$, and $V_A = -0.66 + (0.5 \cdot 1.33) = 0$. Consider, too, blocking. Here Stage 1 A+ contingencies would result in A gaining an associative strength of 1λ. In Stage 2 AB would only need to acquire an associative valence of 0.5λ to reach a net activation level of 1λ since it would generalise half of its associative strength (i.e. 0.5λ) from A. At test the unreinforced element B would generalise half of the associative strength from AB+ and, since $V_B = 0 + (0.5 \cdot 0.5)$, would have an associative strength of 0.25λ. Compare this to no A+ pre-exposure. Here AB+ would acquire an associative strength of 1λ, which means that at test B would, again, generalise half its associative strength from AB, which in this instance would lead to B having an associative strength of 0.5λ. The model, therefore, correctly anticipates that responses to unreinforced element B would be retarded following A+ pre-exposure training relative to no A+ pre-exposure. Pearce's model therefore overcomes some of the disadvantages of strictly elemental associative theories by allowing only a proportion of the associative strength of stimuli not actually present to generalise to present stimuli, whilst still being able to predict many of the empirical phenomena that elemental cue-competition models were developed to account for. One could argue that unique-cue theories may better explain learning phenomena, since they are able to learn any discrimination by the mere addition of a unique cue to a stimulus configuration. On the other hand, Pearce's (1987, 1994, 2002) theory has the benefit of parsimony and of being able to make unambiguous predictions. One of the problems of unique-cue theories is the question of when or if unique cues are added to stimuli. Should a unique cue be added to *all* stimuli, to just compound stimuli, or merely to compound stimuli in the event of a linearly insoluble problem? As we shall see in later chapters this is a real drawback to unique-cue theory since it renders many predictions equivocal and therefore difficult to test.

On the other hand Rescorla (2003) points out that Pearce's model cannot, as it stands, deal with summation, or the observation that responses to an AB compound is stronger than are responses to its elements following A and B preconditioning. The model, however, can deal with summation in a similar way to unique cue theory: by assuming that the experimental context constitutes a common unreinforced element in all stimuli (see Pearce, 1994, also Pearce, Adam, Wilson, & Darby, 1992, and Rescorla, 2003). Similarly the model cannot, as it stands, predict Latent Inhibition, or the relatively slow conditioning of a previously presented, but unreinforced CS. Pearce (1994) suggests that the saliency parameter α be reduced for stimuli in the absence of reinforcement, which achieves the desired prediction of slower conditioning for previously unreinforced stimuli and thereby allows the model to correctly anticipate latent inhibition.

Pearce's (1987, 1994, 2002) theory, therefore, tackles some of the problems associated with elemental theories. There are, however, still problems concerning the extent of generalisation between stimuli. Shanks, Darby, and Charles (1998), for instance, conducted a series of experiments aimed at testing the theory's predictions of retroactive interference using the same type of food allergy task as Shanks, Charles, Darby, and Azmi (1998) and outlined in Chapter One. In experiment 2, for instance, participants received AB+, and CD- trials in Stage 1. In Stage 2 the elements of these compounds were revalued to give A-, B-, C+, and D+. In a test stage participants were given unreinforced AB and CD trials. Pearce's theory predicts that, because of generalisation from elements to compounds, at test participants should give more allergy predictions to CD than to AB. What Shanks et al. found, however, was that participants preserved the discrimination between AB+ and CD- at test, showing a resistance to interference beyond that predicted by either elemental or configural theories alone. This suggests an ability to suspend generalisation when necessary that is seemingly irreconcilable with associative learning theory. Rescorla (2003) came to much the same conclusions following experiments with rats and pigeons. In a series of experiments Rescorla (2003) tested the predictions of both elemental and configural models on a two-stage biconditional discrimination of stage 1 AB+, CD+, followed by test stage AD and BC compounds. His results showed that responses at test were greater for the conditioned AB and CD compounds than for test compounds, thus conforming to the predictions of configural theories, as generalisation between elements was clearly not perfect. However responses to test compounds AD and BC were greater than for their elements, as predicted by elemental theories. Rescorla's results therefore give support to both elemental and configural theories; indeed he ends this paper by calling for a 'principled description of their relative contributions' (Rescorla, 2003, p. 175). The inescapable conclusion is, however, that it is difficult to see how associative learning theories can be adapted to provide consistently accurate a priori predictions without the modeller's intervention. Wagner (2003) has recently proposed one solution as 'context sensitive elemental theory'. Fundamentally this view suggests that if two stimuli are from different modalities (e.g. light, tone) they will be treated configurally in that generalisation will be computed on the basis of an algorithm similar to Pearce's (1987, 1994, 2002). On the other hand, if stimuli are from the same modality, they will be treated elementally and generalisation is computed on the basis of summation in a similar way to the Rescorla-Wagner (1972) model. Wagner (2003) demonstrates the ability of this theory to explain animal conditioning data but in the present context of human conditional learning one has to be sceptical, since the stimuli of Shanks, Charles, Darby, and Azmi (1998) can clearly be viewed as being from the same modality in that they are all foods and yet these data suggest that people solve Human Conditional Learning (HCL) problems configurally.

It seems that responses to stimuli may be mediated by both elemental and configural processes, and that organisms are flexible enough in their approaches to deploy either strategy in order to predict what will happen in their environment. Some theorists take a slightly different approach to modelling learning in that their models incorporate algorithms that generate elemental and configural strategies in parallel, and are grounded in the notion that different types of processing occur in physiologically distinct areas of the brain.

2.2 Neuropsychological Models

Ultimately, all neural network models are neuropsychological in that they are assumed to reflect the way in which connections, or associations, between neurons strengthen and weaken with experience. The models discussed so far, however, do not differentiate between the contributions of discrete neural areas, and therein lies the distinction. This section will discuss what one might term modular models of learning, first describing the models and then reviewing the research evidence that underlies them.

Although by no means the first to promulgate such a notion (for a comprehensive discussion of the history of neural network models see Ellis & Humphreys, 1999), David Marr (1970, 1971) made a seminal contribution in laying the foundations for the models discussed below. He proposed that physiologically distinct brain areas underlay different cognitive processes. Broadly he suggested that the hippocampal region was a fast, but temporary store for memory based on the establishment of associations between coactive neurons. He also suggested that the hippocampal area was an intermediate store between short and long term memories, and that memories stored in the hippocampal region were passed to cortical areas for long-term storage and integration with existing memories. The hippocampus was also, according to Marr, an autoassociator; meaning that the structure could recreate prior patterns of activation following input of a partial stimulus configuration. This allows the structure to contribute to retrieval processes by recreating previously seen stimulus configurations based on partial input. Although the details of Marr's theory may be questioned his broad assertions concerning the function of hippocampal and cortical areas in learning and memory have been largely accepted (c.f. Treves & Rolls, 1992; Hasselmo, 1995). Eichenbaum, Otto, and Cohen (1994) came to similar conclusions: that cortical regions maintain specific representations in the short term, that the parahippocampal region holds individual items in the intermediate term, and that the hippocampus itself forms associations between individual items in different sensory modalities to form a synthesised whole and that eventually these synthesised representations are stored as long term memories in the cortex. Effectively the hippocampus forms a distinct representation that draws together individual details of a memory into a whole. For instance the sight, sounds, smells, tastes, and touches involved in two different memories may be similar, but their conjunction or context makes them unique and separable from one another.

In applying these ideas to associative learning Rudy and Sutherland (1989, 1995; Sutherland & Rudy, 1989) suggested that learning new associations is contingent upon two processes taking place in two physiologically distinct brain areas. Elemental, or simple, associations may be formed in cortical areas whereas configural associations are dependent on the hippocampal and medial temporal lobe areas, although these representations are ultimately available to cortical areas and are stored there long term. Simple associations, in this view, are formed on the basis of error correction rules, such as that specified in the Rescorla-Wagner (1972) model. When circumstances make a linear solution impossible the hippocampal system differentiates representations of similar stimuli leading to different outcomes, allowing the solution of such tasks as the NPP or biconditional problem (Rudy et al. 1995). This differentiation of similar stimuli is proposed to be the result of hippocampal 'binding' of complex stimuli to form an integrated and unique stimulus representation. This is similar to configural theory, but without the assumption that generalisation between elements and compounds is inevitable, since organisms can learn to differentiate between times when generalisation is helpful, and when it is not. Gluck and Myers (1993; 1996; 2001) proposed a similar model, which, since the models make comparable predictions, will be examined in more depth.

In the Gluck et al. (1993) model associative learning is again split between two modules representing cortical and hippocampal function. The model formalises the idea that cortical areas, such as the frontal lobes in contingency learning, cannot create configural representations, but make behavioural predictions on the basis of simple elemental, or stored configural, stimuli. According to both Gluck et al. (1993) and Rudy et al. (1995) the hippocampal area is vital for the formation of internal representations of new configurations of elements, or stimulus-stimulus learning. Put more prosaically, cortical areas are seen to be responsible for stimulus-response learning whereas the hippocampal region is responsible for stimulus-stimulus learning. Both models make the broad suggestion that learning reliant on elemental assumptions is spared following hippocampal damage, since learning to respond to existing or elemental stimuli is the province of frontal areas. The formation of associations between CS and US is therefore postulated as frontally mediated, whereas the configuration and representation of CSs is envisaged as a hippocampally mediated function.

The Gluck and Myers (1993) model comprises two separate modules intended to represent the functioning of the frontal and hippocampal areas. This is reflected in the model's architecture. The frontal module converges to a single outcome, and generates predictions of US presence. The hippocampal module recreates the input at its output nodes, and takes account of the presence or absence of the US. Both modules are based on three layer connectionist architectures and update their weights according to an error backpropagation algorithm (Rumelhart, Hinton, & Williams, 1986). The inner layer of the hippocampal module contains fewer nodes than the input layer. This allows the hippocampal layer to perform redundancy compression based on feedback concerning the presence or absence of the US. If two stimuli both predict similar outcomes then the model will compress their representations. This reflects the internal architecture of the brain since the medial temporal region receives multimodal sensory input into the entorhinal cortex, which has far fewer outputs than inputs. If, however, two inputs lead to different outcomes their representations will be separated, a process Gluck and Myers (1993) call predictive differentiation. The major difference between the two modules is that the hippocampal module can perform redundancy compression and predictive differentiation, whereas the frontal module cannot. Representations can, however, be transferred from the hippocampal to the frontal module where they can be stored in the long term used to predict the presence or absence of the US.

The model predicts, therefore, that hippocampal region damage results in an inability to form new configural representations, since the frontal module alone works on elemental assumptions and cannot distinguish between similar stimuli with different outcomes.

These assumptions are specified according to the following set of equations. Both modules first calculate their output before updating their weights, as illustrated by Equation 2.6.

Equation 2.6: $V_j = \Sigma o_i \cdot w_{ij} + \theta_j$

Here the activation of internal layer node *j* (V_j) on any given trial equals the sum of the outputs of the input layer (Σo_i) multiplied by the weight of the connection between the input layer and the inner layer (w_{ij}) plus a bias term (θ_j), representing the tendency of that node to become active. The output of the inner layer (o_j) is then calculated with Equation 2.7, a sigmoid function that reduces the overall activation of node *j* (V_j) to a number between 0 and 1. The activation of output node *k* is then calculated according to Equation 2.6. This constitutes the model's predictions for the presence of the US for both frontal and hippocampal modules, and the recreation of inputs for the hippocampal module alone.

Equation 2.7: $o_j = \dfrac{1}{1 + e^{-V_j}}$

Having calculated the output for both modules the next step is to calculate the error (δ_j) between the model's predictions and the US or input recreation at the output layer. This is achieved using Equation 2.8, where the desired outcome (or US: $\boldsymbol{d_j}$) is compared with the model's prediction (or actual output: o_j) and multiplied by a function of the activation of node j ($f'(V_j) = V_j - (1 - V_j)$).

Equation 2.8: $$\delta_j = (d_j - o_j)\, f'(V_j)$$

The next stage represents one of the glaring assumptions of the error backpropagation model. The model proposes that internal layer error (δ_i) is a function of the outer layer error (δ_j), as Equation 2.9 shows. Theoretically the internal layer is meant to reflect internal representations of stimuli, although this view is hardly universally accepted. Although the idea that error can be 'backpropagated' from the internal layer to the input layer provides a neat engineering solution to the problem of differentiating the representations and outcome of similar stimuli there are no real physical correlates of this process and this is a major drawback, along with the fact that the backpropagation process takes many more trials to acquire even simple discriminations than more straightforward elemental or configural models of learning.

Equation 2.9: $$\delta_i = (\Sigma \delta_j \cdot w_{ij})\, f'(V_i)$$

Having calculated the error for both internal and output layer nodes the weights for both sets of connections between nodes are updated according to Equation 2.10, which is simply a variant of the competitive error correction rule used in the Rescorla-Wagner model.

Equation 2.10: $$\Delta w_{ij} = \beta\delta_j \cdot o_j$$

So, how accurately does the model reflect both physiological and behavioural data? To assess this it will be necessary to examine what is known about the cognitive function of frontal and hippocampal areas, and to relate this evidence to the Gluck et al. (1993, 1996, 2001) model to build a picture of which processes may be involved in associative learning, and their likely neural bases.

2.3: The Hippocampal Region

That the hippocampal region is involved in learning and memory is not in doubt, rather the debate surrounding this area concerns the precise details of mapping specific structures to particular functions. Physically the hippocampus is subsumed into the larger region of the medial temporal lobe, but the function of this broad area is often attributed to the hippocampus alone. Signals flow, roughly unidirectionally, towards the structure from all modalities, converging on the entorhinal cortex, which is recurrently connected to cortical areas. Some signals are then transmitted straight to the hippocampus proper, whereas others enter via sparse but strong activation from the dentate gyrus. Signals then pass through the hippocampus before being transferred back to cortical areas via the subiculum and entorhinal cortex. Many authors use the term ‘hippocampus’ to describe the general area and surrounding structures. Gluck et al. (1993) are no different in this regard: the theory explicitly states that the model describes broad hippocampal region processing, rather than the hippocampus *per se*. One problem this creates is sometimes contradictory observations in the literature contingent on specific sites of damage. For example Jarrard (1993), found no decrement for negative patterning problems in rats with lesions confined to the hippocampus proper, compared to Sutherland et al. (1989), who found disruption to rat's negative patterning learning following broad hippocampal region lesions.

Scoville and Milner (1957) used the term 'hippocampal region' to describe the extent of damage to the patient HM who had undergone bilateral surgery to remove the hippocampus, subiculum, and the amygdala. In addition HM's surgery, intended to ameliorate severe epilepsy, damaged the entorhinal, and perirhinal cortices (collectively referred to here as parahippocampal areas). HM's short-term memory was intact, as were details of his past in long-term memory. What his case, and those of others with medial temporal lobe damage, has shown is that the region is involved in the formation of new episodic memories, or explicit memories for events integrating information from several sensory modalities. Put another way Scoville and Milner (1957) came to the conclusion that damage to the hippocampal region resulted in anterograde amnesia, or the inability to form new memories. Some researchers pointed out, however, that surgical procedures were imprecise, and that damage extended beyond the hippocampal region. For instance Mishkin (1978) found that damage to both the hippocampal area and the amygdala was necessary to produce episodic learning difficulties in monkeys. Subsequent research (e.g. Squire & Zola-Morgan, 1985) showed that hippocampal lesions alone in monkeys could produce statistically significant memory deficiencies, although damage to the amygdala exacerbated these differences. Another distinction was formed as a result of HM and other patients' abilities to learn new sensori-motor skills and perform normally in implicit memory tasks, such as word fragment or pattern completion (Eichenbaum et al. 1994; Squire, 1992). Hippocampal region damage seems to disrupt only episodic, conscious memory. Importantly, for Gluck et al. (1993), the formation of a Conditioned Response (CR) is unimpaired following hippocampal damage (Gabrielli, McGlinchey-Berroth, Carillo, Gluck, Cermack, & Disterhoft, 1995), as is simple category learning (Knowlton, Squire, & Gluck, 1994). This fits in with the idea of CS-US associations being independent of hippocampal processing and suggests that contingencies may be processed in cortical areas.

Zola-Morgan, Squire, and Amaral (1986) reported the case of RB, a patient who experienced severe memory dysfunction following a difficult heart bypass operation. Histological examination revealed that RB's damage was restricted to bilateral lesions of the hippocampi. Functionally RB was, along with three amnesics, and a group with Korsakoff's syndrome, compromised relative to healthy and alcoholic control groups on the Wechsler Memory Scale, paired associates learning, story recall, and diagram recall, suggesting again a problem with relationships between elements in memory. Additionally there was little evidence for any retrograde amnesia, or forgetting of past events for R.B., and he performed above normally on the Wechsler Adult Intelligence Scale, apart from the Digit Symbol Substitution Test that requires the explicit association of numbers with symbols in memory for success. Despite this RB still outperformed HM, who had more broad bilateral lesions to the whole medial temporal lobe, again suggesting that the extent of damage has some relation to the extent of impairment.

Studies using fMRI and PET scans yield comparable conclusions. Cohen, Ryan, Hunt, Romine, Wszalek, & Nash (1999) suggest that the hippocampal region processes the relations between multimodal memory elements automatically, although strategic intervention can influence this process. The idea that stimulus elements can be bound together and that further strategic, presumably frontally mediated, processing can influence the process is analogous to Gluck and Myer's (1993) compression and differentiation processes, respectively. Cohen et al. (1999) also suggested that the hippocampal region is implicated in both encoding and retrieval. In a review of research on sufferers of medial temporal lobe epilepsy Baxendale (1995) compares MRI assessed hippocampal volumes to performance on various neuropsychological tests. Again she came to the conclusion that the hippocampal formation is implicated in binding memories, and emphasises the laterality of function in that right hippocampi tend to be involved in spatial tasks, whereas left hippocampi tend to be involved in verbal tasks, such as paired associate learning. Additionally Henke, Weber, Kneifel, Wiesler, & Buck (1999) found hippocampal involvement in establishing semantic relations between words by PET scan, again both assessments suggest the area's involvement in establishing relationships between concepts, objects, or stimuli.

Recall that the hippocampal region forms new representations, according to Gluck and Myers (1993), by performing two functions: redundancy compression and predictive differentiation. Redundancy compression refers to an increase in generalisation between reliably co-occurring or similar stimuli. This function results in a compressed representation of input stimuli and facilitates the learning process by ensuring that processing resources are not wasted on non-salient stimuli. Myers, Gluck & Granger (1995) suggested that this function is performed in the parahippocampal, entorhinal, and perirhinal cortices on the basis of stimulus-stimulus regularity. This hypothesis is biologically plausible as there are many more inputs into the entorhinal cortex than outputs to the hippocampus (Myers et al. 1995), implying that more information enters this region from the cortex than is outputted to the hippocampus. Predictive differentiation refers to a decrease in generalisation between stimuli that reliably predict different outcomes and is suggested to occur in the hippocampus proper (Myers et al. 1995). Hopkins, Myers, Shohamy, Grossman, and Gluck (2004) have advanced more recent evidence that participants with MRI verified hippocampal damage are compromised in terms of probabilistic category learning tasks. In this instance a weather prediction and 'ice cream' task was used where participants had to predict if the weather would be rainy or sunny in the former instance, or whether a customer wanted vanilla or chocolate ice cream in the latter instance. In the weather prediction task, stimuli were one of four types of cards that predicted sunny weather with a probability of 0.8, 0.6, 0.4, and 0.2. The ice cream task was identical in logical structure, but used four different faces made from a Mr. Potatohead™ set (for those unfamiliar with Mr. Potatohead, 'he' is a patented child's toy comprising a potato shaped 'body' to which various body parts can be attached. Hopkins et al. (2004) provide pictures of stimuli). In both cases stimuli could be presented alone or in combination. Hopkins et al. (2004) found that patients' learning was compromised relative to controls in both tasks. Furthermore, it was found that hippocampal damaged participants used simple strategies involving single cues whereas controls used more complex multi-cue strategies. Again, this fits well with the idea that the hippocampal region does not mediate simple stimulus-response learning but becomes involved when multiple cues are present and may be involved in stimulus-stimulus compression or predictive differentiation. While the suggestion that the hippocampus mediates these two functions is therefore an admirable account, there are problems here, however, in accounting for Jarrard's (1993) results for preserved negative patterning learning in restricted hippocampal damaged rats. Presumably solution of this problem would require both functions, and while this account has many advantages it is probably prudent at the present time to restrict any assessment to broad hippocampal or medial temporal lobe region rather than looking at specific anatomical functions.

Notable in its absence thus far is any mention of the influence of neurotransmitters. Myers, Ermita, Harris, Hasselmo, Solomon, & Gluck, (1996) suggest cholinergic modulation of hippocampal function through the medial septum. Acetycholine has been characterised as a 'neuromodulator' that has several important effects, including suppression of synaptic transmission (Hasselmo, & Schnell, 1994). When acetylcholine is absent the hippocampal structure's recurrent collateral cells are activated. These cells have a high degree of internal connectivity: the property that allows pattern completion and autoassociative functions on the basis of correlation of activity (c.f. Marr, 1970, 1971; Treves & Rolls, 1992; Hasselmo, 1995). During this type of activation hippocampal EEG shows the sharp, non-rhythmic bursting activity (Fox, Wolfson & Ranck, 1983) associated with consummatory behaviours such as eating and drinking (Vanderwolf & Leung, 1983) and analogous to a recall or consolidation mode. In this case the hippocampal region may be performing pattern completion: retrieving a whole memory, or stimulus, from partial input. By contrast an increase in hippocampal acetylcholine via connections in the medial septum results in selective suppression of internal recurrent collateral cells (Hasselmo, Schnell, & Barkai, 1995). This suppresses the autoassociative function of the hippocampus by preventing activation spreading to other potentially co-active cells. Suppression of autoassociation may also be a means of preventing catastrophic interference (c.f. Marr, 1970, 1971): the confusion resulting from a partial input activating too many different configural representations. This regular activity allows new representations to be stored (Buszaki, 1989), and is associated with regular theta wave (4-8Hz.) activation (Fox et al. 1983), and exploratory behaviours (Vanderwolf et al. 1983). Cholinergic disruption could account for evidence that medial septal lesion results in disrupted hippocampal function in rabbit eyeblink studies (e.g. Salvatierra & Berry, 1989). On the other hand studies using scopolamine (a cholinergic antagonist) on rabbits (Solomon & Gottfried, 1981; Solomon, Groccia-Ellison, Flynn, Mirak, Edwards, Dunehew, & Stanton, 1983) and humans (Solomon, Solomon, Van der Schaff, & Perry, 1993) have shown similar disruption to hippocampal function. These latter studies are more specific to cholinergic, rather than medial septal function and suggest that acetylcholine may have a role in modulating storage and recall functions in the hippocampus.

To summarise: there is good evidence of hippocampal region, or medial temporal lobe, involvement in learning and memory, although there is too little evidence to be able to say with any certainty what the specific functions of particular components of the system are. As a whole the region seems to be responsible for encoding and retrieval of relationships between multimodal components, or stimuli, within episodic memory. This fits well within Gluck and Myer's (1993) conception of a system that compresses stimuli that predict similar outcomes. Interactions with strategic processing centres in the frontal lobes may produce the suggested predictive differentiation function. Storage and retrieval functions may be mediated by acetylcholine via projections from the medial septum and a lack of the neurotransmitter may result in reduced ability to store new memories and to inhibit autoassociative functions, perhaps leading to interference in memory.

2.4: The Frontal Lobes

Gluck et al. (1993) suggest that frontal regions mediate the stimulus-response relationship, whereas the hippocampal region mediates stimulus-stimulus relationships. So far we have seen the theory offers a reasonable account of the contribution of the medial temporal lobe region to associative learning. The question remains as to whether their theory offers a reasonably compelling account of the contribution of the frontal cortex. To assess this it will be necessary to examine what other researchers and theorists suggest the function of this area is.

There are a number of cognitive functions associated with the frontal lobes. For instance Goldberg and Bilder (1987) suggest that the prefrontal lobes are implicated in the formation and execution of internally generated plans and strategies, the executive direction and control of cognitive processes, attention, memory, and planning.

Certainly this view is consistent with the evidence gathered by Shallice and Burgess (1991). They asked FL patients to buy specific items and obtain particular information from particular shops in a shopping centre in a pre-determined sequence at specified times. Their problems completing the task were unrelated to simple memory for actions, but rather reflected difficulties formulating a plan of action on their own initiative and completing a complex sequence of behaviour. Furthermore patients with frontal lobe damage seem unable to learn from past experience and use this information to plan future actions (Bechara, Tranel, & Damasio, 2000). It is clear that a decrement in the ability to use past experience to guide future behaviour may be related to associative learning abilities, and to the error correction mechanism proposed by Gluck.

In addition many researchers (e.g. Miyake, Friedman, Emerson, Witski, Howerter, & Wager, 2000; Damasio, 1994) have found that patients with frontal lobe damage are impaired at laboratory tasks that involve planning and executing a series of cognitive operations, such as the Tower of Hanoi task in which a set of disks must be moved from one configuration to another on a pegboard according to a set of rules. Similarly FL patients tend to become confused between task relevant and irrelevant stimuli and have difficulty in consciously differentiating between stimuli with different outcomes (Henkel, Johnson, & De Leonardis, 1998; West, 1996; Dimitrov, Granetz, Peterson, Hollnagel, Alexander, & Grafman, 1999), and the appropriate allocation of processing resources in WM (Hartman, Pickering & Wilson, 1992).

Another task that seems sensitive to frontal lobe damage is the Wisconsin Card Sorting Task (WCST: Shallice et al. 1991; Dempster & Corkhill, 1999; Miyake et al. 2000). Here participants must sort cards into groups according to an unnamed rule that is periodically changed. Frontal lobe damage is indicated by participants' initial difficulty in acquiring rules as well as in their perseverance with old rules that no longer apply: this type of mistake is called a perseverative error. Furthermore Gunning-Dixon and Raz (2003) found that prefrontal cortex volumes predicted the number of WCST errors, that age-related shrinkage of the pre-frontal cortex has been associated with an increase in perseverative errors (Raz, Gunning-Dixon, Head, Dupuis, & Acker, 1998). These data suggest that patients with frontal lobe lesions seem to have some difficulty in discriminating between valid and invalid responses, and responding flexibly to environmental changes. This certainly seems consonant with Gluck's view of the role of the frontal lobes as an error correction centre involved in the conscious prediction of the consequences of stimuli.

A further task associated with the frontal lobes is the Stroop (1935) task: participants must name the ink colour of a series of colour words printed in either colour congruent or colour incongruent ink. Typically all participants show a performance decrement when naming the ink colour of words printed in colour incongruent ink relative to naming the ink colour of words printed in colour congruent ink; but participants with frontal lobe damage show a greater deficit (e.g. Perret, 1974; Damasio, 1994). The standard interpretation of Stroop task deficits is the inability to withhold, or inhibit, inappropriate responses. Certainly this is consonant with the inappropriate affect and social responses exhibited by frontal lobe patients (Damasio, 1994). It may also be consistent with Gluck's suggestion that the frontal lobes mediate stimulus response formation in that the solution of a learning task requires the suppression of inappropriate responses to stimuli. There are, therefore, some compelling data from analogous tasks to suggest that Gluck's hypothesis of frontal lobe involvement in the acquisition of stimulus response associations may be a reasonable assumption.

There are neuropsychological, animal lesion, psychopharmacological, and imaging studies that support Gluck's hypothesis. Rolls (2000, 2004) suggested that the orbitofrontal cortex is implicated in representing the reward value of a stimulus and clearly this is related to learning about outcomes.

Furthermore this area is reciprocally connected to the entorhinal cortex, which is in turn reciprocally connected to hippocampus, forming a physiological basis for Gluck's theory that similarity between stimuli is attenuated by feedback about the presence or absence of the US. Evidence for this comes from human fMRI studies that show an increase in activation within this area by pleasant or painful, rather than neutral, stimuli regardless of stimulus intensity (Francis, Rolls, Bowtell, McGlone, O'Doherty, Browning, Clare & Smith, 1999), suggesting that frontal areas are implicated in assessing the consequences, or reinforcement value, of environmental stimuli. Similarly Rolls (1999) found a hunger dependent increase in electrical stimulation within this region on presentation of food reward in primates.

Furthermore there is evidence that orbitofrontal regions are implicated in learning associations between stimulus and reinforcer representations: primates with lesions to the orbitofrontal cortex continue to respond to no longer reinforced stimuli in extinction studies (Thorpe, Rolls, & Maddisson, 1983). This deficit is consistent with the idea that frontal regions have a part to play in mediating appropriate responses to stimuli.

Another paradigm used to investigate the role of frontal areas is the discrimination reversal task in which the consequences of two stimuli are reversed. Here both monkeys (e.g. Thorpe et al. 1983) and humans (Kringelbach, O'Doherty, Rolls & Andrews, 2003) showed orbitofrontal activation in response to contingency reversals. In the former study specific neurons in monkeys' orbitofrontal cortex responded to a blue stimulus when rewarded with a glucose compound but not to a green stimulus associated with a saline drink. Following reversal to a green stimulus associated with the reward and to a blue stimulus associated with a saline drink inhibited response of formerly active neurons. In the latter case a human face discrimination task was employed in which two faces were displayed at the same time and participants had to choose between them. A correct response resulted in a happy face, whereas an incorrect response resulted in an angry face: part of the orbitofrontal cortex was activated more during a discrimination reversal relative to a control condition. These data seem to indicate that learning about the consequences of actions and responding quickly and accurately to changes in the environment may well be the result of processing occurring within frontal areas of cortex.

One further strand of evidence comes from neuropsychological studies of paired associate learning. For instance when participants are given an experimental paired associates test for recognition, free and cued recall of semantically related and unrelated pictures and words it is those with frontal lesions that are impaired relative to a normal control group (Dimitrov, Granetz, Peterson, Hollnagel, Alexander, & Grafman, 1999). This pattern of deficits is greater for free recall than cued recall, and least for tasks involving recognition recall. There is also a bigger deficit for unrelated relative to related words. This implies again that the frontal lobes may be involved in associative learning, although in this particular study deficits in paired associate tasks were exacerbated by atrophy of the temporal lobe. This is hardly surprising since paired associate tasks presumably tap into both stimulus-stimulus and stimulus response learning.

In the context of Rolls' (2000, 2004) work and Gluck's theory, however, it is clear that a standard paired associate task does not easily fit into a framework of stimulus associations with outcomes or contingencies, since there are no clearly identifiable outcomes in paired associates studies. It is very difficult, therefore, to identify separable stimulus-stimulus and stimulus-response associations in paired associate tasks, and these tasks may involve both processes and therefore both frontal and medial temporal areas.

The neuropsychological evidence points, therefore, to a reasonable amount of support for the Gluck and Myers (1993) cortico-hippocampal model. The medial temporal lobe generally, and the hippocampus in particular, does seem to play a part in mediating stimulus-stimulus associations, whereas cortical areas in general, and frontal areas specifically, do seem to be involved in creating stimulus-response associations. This model, however, makes very broad assumptions about the relative contributions of each area, whereas the evidence suggests a more complex picture in which the notions of 'hippocampal' and 'frontal lobe' contributions constitute gross overgeneralisations.

In reality it is likely that explanations of the function of both these regions will have to be subdivided into more detailed descriptions of the individual and combined contributions of smaller groups of neurons. Furthermore, the involvement of frontal areas of the brain seems, given the evidence, to be more extensive than Gluck's theory assumes in that Gluck merely supposes that cortical areas learn stimulus-response associations, whereas the evidence suggests that the frontal lobes are also involved in the inhibition of automatic responses and therefore should be capable of modulating learning.

Gluck et al. (1993) assume that the hippocampus is the only structure that modulates learning through predictive differentiation and stimulus-stimulus compression, and while this may be it is clear that cortical areas have a part to play here too. One further major caveat to the Gluck model in particular, and any models similarly based on the error backpropagation algorithm (Rumelhart et al. 1986) in general: the inability to predict one trial overshadowing. The error backpropagation algorithm generally takes hundreds, or sometimes thousands of trials to learn even simple discriminations, but empirical observation shows behavioural changes after only a single trial (Pearce, 1994).

The modular nature of Gluck et al.'s (1993) model can also be regarded as inelegant, unparsimonious and rather unwieldy in comparison to configural or elemental models of learning, and the model itself makes assumptions about representation and the neural bases of generalisation that may be described as tenuous at best. It may, therefore, be more prudent to focus on the distinction between elemental and configural models in determining how participants generalise between stimuli in contingency learning. Although the sections on the hippocampus and frontal lobes may now seem irrelevant it was important to consider the role of these areas in associative learning since, as shall be seen in subsequent chapters, these two brain areas seem particularly prone to the adverse effects of the ageing process.

Clearly, though, none of the models of learning discussed so far are the 'last word' in learning theory. All of the extant models have weaknesses and strengths but there are some empirical phenomena that remain unpredictable. One such observation concerns rule induction in NPPs and PPPs. Shanks and Darby (1998) reasoned that it was possible to solve both of these problems concurrently by adopting a rule of conjunction: elements and compounds predict opposing outcomes. Using the same food allergy paradigm described earlier they found that participants who learned contingencies quickly and thoroughly gave responses consistent with rule induction. For instance in experiment 1 participants were given A+, C-, E+, G-, and I+ trials in stage 1, followed by B+, D-, F+, H-, and J+ contingencies in stage two followed by AB-, CD+, EF-, GH+, and KL- discriminations in stage three. At test participants were tested on all seen elements and compounds, and, critically, the novel compound IJ and unseen elements K and L. They found that participants who learned the initial discriminations more slowly tended to respond to the critical IJ, K, and L trials by summation: giving IJ+, K-, and L- as solutions. Participants who learned the initial discriminations quickly, however, exhibited a very different pattern of responses. They seemed to generalise a rule of conjunction and gave IJ-, K+, and L+ responses at test.

Whilst it is a comparatively simple matter to state that people who learn problems well may abstract generalisable rules to aid future solution of similar problems, specifying this rule mathematically in a model of learning is a far from trivial matter. It is difficult to see how rule induction can be incorporated into computational models of learning since it is almost impossible to predict *a priori* what rules will be induced when, or how far they will be generalised to which other situations.

Another problem is that of the precise nature of the rules induced in Shanks and Darby's (1998) studies, although parallels with the problem solving literature that may help to illuminate this area. In associative learning there is a phenomenon known as Easy-Hard transfer in that learning an easy discrimination can aid a harder discrimination between similar stimuli. Lawrence (1952) first demonstrated that pigeons trained on an easy discrimination between a dark and light grey later mastered a more difficult discrimination between two mid greys quicker than pigeons that had been exposed to the harder discrimination all along. This phenomenon has been replicated in rats (Gluck & Myers, 1993, 1995) and in humans with a face discrimination task (Suret & McLaren, 2003). Furthermore pre-training with an easy problem prior to discrimination reversal also facilitated learning in a subsequent hard problem relative to trials involving the hard problem itself in pigeons (Mackintosh & Little, 1970) and humans (Suret et al., 2003). Curiously enough there have, although few, been empirical demonstrations of Easy-Hard transfer in the solution of insight problems. These can be characterised as involving a sudden restructuring of the way participants conceive of a problem and its solution (Weisberg, 1996), based on a gradual accumulation of knowledge involving effortful, directed thought (Novick & Sherman, 2003) and it is certainly a possibility that the rules induced by Shanks and Darby's (1998) participants may follow this pattern.

Weisberg and Alba (1981), for instance, found that presenting participants with easier analogues of the nine dot and triangle problems facilitated performance on the tasks themselves whereas verbal hints did not, suggesting that direct experience of a problem within the same domain resulted in transfer of a solution. Novick and Holyoak (1991) demonstrated that solving analogical, easier mathematical problems resulted in better performance than if participants were simply allowed to attempt the more difficult problems alone. More pertinently Luo and Niki (2003) recently demonstrated that the solution of Japanese riddles led to greater right hippocampal activation in young participants together with broad neocortical activity, including within the frontal regions. All of which suggests that obtaining an insight into the solution of a problem may be contingent on knowledge of how to solve the problem itself, or similar problems, and is contingent on an interaction between hippocampal and cortical regions.

There is no reason to suppose that rules induced during an HCL experiment should be any different since they, too, seem to be contingent on initial learning involving an interaction between hippocampal and cortical areas.

In conclusion the models of learning discussed in the previous two chapters have developed in sophistication and accuracy considerably over the last eighty years or so, and much progress has been made in terms of our ability to predict learnt responses in both animals and humans. In parallel with this effort many of the neural bases of learning have been identified and some of this understanding has been expressed in neuropsychological models of learning. While this progress has been considerable, there is much work to be done before we can truly say that that we actually understand and can predict how learning occurs and what the neural bases are, even in relatively simple species such as rats.

The focus of the present research is not, however, in developing new models of learning but on the effects of ageing on our contingency learning ability, although the data may illuminate some theoretical aspects of learning. To be able to make predictions from existing learning theories in the absence of extant data specifically concerning age effects on conditional learning it will be necessary to look at both general and specific theories of cognitive ageing and to extrapolate our predictions from what is known about learning and what is known about cognitive ageing.

Chapter 3: Introduction to Cognitive Ageing

3.1: Rationale

Research into the cognitive abilities of older people has become increasingly important in recent years. Older people now constitute a large and rising proportion of the populations of developed countries. The 2001 census found a greater number of over 60's (21%) than under 16's (20%), and the trend for an increasing proportion of elderly and a diminishing proportion of young people is predicted to continue (Office for National Statistics, 2002). This has resulted in an increasing focus on research into the effects of ageing on cognitive ability. Among the questions this raises is what and how much can we expect older people to adapt to change and learn new skills?

3.2: Early Ideas

Early studies were somewhat pessimistic in their findings and conclusions concerning cognitive ability in old age. For instance Yerkes (1921) found a marked and monotonic age related decline in participants' performance in Army Alpha I.Q. test scores, beginning in their thirties and continuing progressively and systematically through the rest of the lifespan. Similarly Wechsler (1955, 1958) found a comparable pattern in the overall standardisation data for the Wechsler Adult Intelligence Scales (WAIS), and most early research seemed to reflect this (see Salthouse, 1991; Birren & Schroots, 2001 for reviews). This rather bleak outlook may, however, be misleading for methodological, empirical, and theoretical reasons.

3.3 Methodological Considerations

One major problem with the data from these studies is that they are all derived from cross-sectional research. This effectively means that one is comparing two separate age groups, or cohorts, of people born and brought up in different times according to their current capabilities. A major problem with this methodology is that different cohorts will have been exposed to different educational opportunities, quality of health care and diets, parenting styles, amount of physical exercise, social activities and any number of other factors and experiences that may affect intellectual ability in later life. One important factor, for instance, in nearly all cross-sectional research carried out in the last half century is the fact that every member of an ageing sample had lived through the Second World War, whereas younger participants had not. The privations of war, rationing, and an interrupted education may have had effects on the physical and cognitive development of those who experienced them that are almost impossible to quantify or control for. Indeed Flynn (1984, 1987) found that average I.Q. scores had increased generation-by-generation, birth year on birth year, suggesting a systematic increase in cognitive ability for exogenous, rather than endogenous reasons. The implication of this is that cross-sectional differences in general intellectual ability are nothing to do with ageing *per se* but rather reflect improvements in the living conditions, education, diet, and healthcare experienced by succeeding generations. On the other hand, even if one accepts the view that cross-sectional research is fundamentally flawed and inaccurate as a result of cohort effects one could argue that it may still be useful in describing differences in cognitive ability between existing cohorts, although any observed differences could potentially be exaggerated.

The obvious answer to these problems is to compare ability longitudinally: taking measures from the same cohort of people at different points in their life. Typically results from these studies suggest that general intellectual ability, as measured by IQ tests, tends to be preserved until late middle age at least (e.g. Cunningham & Owens, 1983). Despite this there are still methodological problems with longitudinal studies. For instance, those who drop-out of longitudinal studies tend to be those who do less well on the tests, suggesting that longitudinal studies may minimise age differences (Riegal & Riegal, 1972; Rabbitt, McInnes, Diggle, Holland, Bent, Abson, Pendleton & Horan, 2004). Another problem is that practice may aid older people when they retake tests at subsequent intervals (Salthouse, 1991; Rabbitt et al. 2004): here the inference is that data collected using a longitudinal methodology may underestimate the extent of cognitive decline with age.

Schaie (e.g. 1983, 1986; Schaie & Hertzog, 1983, 1986) adopted a more sophisticated methodology: the cross-sequential method. This involves not only testing the same cohort, or group, of individuals at different points across their lifespan but also recruiting new cohorts at regular intervals. By combining a cross section and longitudinal methodologies Schaie was able to compare both cross-sectionally and longitudinally, and also make time-lag comparisons. Time lag comparisons are made between different cohorts of the same age at different points in time. The assumption is that any differences between two groups of people of the same age at different times must be due to cohort membership, presumably as a result of exogenous factors. His research indicated that cognitive decline in general intellectual ability, as measured by the Primary Mental Abilities battery (PMA: Thurstone, 1958) may be delayed until one's mid-sixties. Furthermore his data appear to confirm the observation that cross-sectional studies exaggerate age differences whereas longitudinal studies minimise them. In a re-analysis of Schaie's (1983) data Salthouse (1991) calculated the extent of time-lag effects on the data. He found that if one controlled for time-lag the differences between longitudinal, cross-sectional, and same cohort data were virtually eliminated. It could be argued, therefore, that cohort effects may be negligible since subsequent generations should still show a qualitatively similar pattern of age differences in cognitive ability. Recall that Flynn (1984, 1987) found a monotonic year-on-year rise in average IQ scores. This too suggests that, since cognitive ability seems to rise systematically cohort by cohort, one can expect to see little difference in the pattern of differences between older and younger people in subsequent generations, and that any specific changes in data will be quantitatively, rather than qualitatively different.

It would be a mistake, however, to ignore cohort effects altogether. The data under consideration were derived from relatively narrow batteries of tests designed to test general ability (Schaie used the PMA, and Flynn analysed WAIS data) and there is no guarantee that when one considers specific cognitive abilities that there will be a similarly monotonic 'Flynn effect'. It does, however, legitimise the use of cross-sectional comparisons in the absence of extant data for longitudinal and cross sequential comparisons, and indicate that such a methodology may provide a good idea of the nature of age differences both now and in the future.

Another methodological consideration concerns the nature of the comparisons being made. Salthouse (2000, 2001) suggested that researchers have taken one of two broad approaches to investigating age differences in cognition. He has labelled these the micro and macro approaches. In this conception micro approaches deal with the investigation of processes underlying specific cognitive tasks that may be impaired with age. Researchers carrying out these types of investigations suggest that decrements in these underlying processes are responsible for any observed age-related differences in the task under consideration. In the case of associative learning, for instance, Pearce's (1987, 1994, 2002) theory, discussed in Chapter Two, suggests that generalisation between stimuli is mediated by perceptual similarity, and that generalisation is a discrete process that underlies the broader ability of associative learning. Any age-related changes in generalisation processes may therefore be responsible for any observed differences between age groups in terms of associative learning performance. Salthouse (2000, 2001) suggests that these kinds of studies and theories can be informative in identifying age-related differences, but that such research does not answer the question of why these processes may decline with age. Such research tends to ignore performance on other tasks and makes the potentially serious mistake of assuming task independence when there is a distinct possibility that age related differences in a range of cognitive tasks could be explained by more general factors. Salthouse himself advocates the use of what he terms macro approaches. This approach takes the view that age-related cognitive decline may be due to more fundamental, general factors reflecting processing resources underlying performance on a range of tasks. For instance, Wechsler (1955, 1958) suggested the standardisation data from the WAIS reflected an overall cognitive decline in general intelligence that underlay age-related cognitive decline in many other tasks. By way of another example, Salthouse (1996) has proposed that poor performance in tests of cognitive ability may be attributed to the general factor of 'processing speed' (this theory will be discussed in more detail in Chapter 4). Again, the suggestion is that cognitive abilities are not independent and can be explained by more general factors that co-vary with age.

3.4: Is 'General Intelligence' an Adequate Conception?

The next issue to consider is that so far only measures of general cognitive ability have been taken into account. This conforms to the idea that a single general factor, or g, underlies all intellectual ability (Spearman, 1927) and that this ability declines monotonically with age, perhaps reflecting an age related physical decline within the nervous system. One major problem with this approach is that some cognitive abilities seem to decline whereas others do not. This appears to be a consistent finding, even in early research. Foster and Taylor (1920), for example, found age differences favouring younger people in rearranging words to make sentences, visual memory, and word fluency whereas older people were superior in verbal abilities. Similarly Flynn (1984, 1987) found greater age-related decline in abstract problem solving than for verbal tasks in his WAIS data. Schaie (1985) found comparable results in his cross-sequential study: age declines were more apparent in reasoning and spatial tasks than the verbal meaning, word fluency, and number tests in the PMA. Earlier theories that suggested an overall and monotonic decline in general cognitive ability with age would therefore seem to be overly simplistic and therefore flawed. Rabbitt (1993; Rabbitt et al. 2004) in an exhaustive review of his and others' data, found much the same pattern of results: participants' scores on the Alice Heim 4 (AH4) group test of general intelligence (Heim, 1968) declined with age, whereas scores on the Mill Hill Vocabulary scale (MHV: Raven, 1982) became better with age. Interestingly Rabbitt (1993; Rabbitt et al. 2004) also observes that while the distribution of scores on the MHV remained constant over the life span the distribution of scores on the AH4 changed over the life span such that with increased age the proportion of participants attaining low scores increased whilst the range of scores remained the same. Put together these data indicate that age-related cognitive decline is not a unitary or global phenomenon, nor is it inevitable for all ageing people, even of the same cohort.

3.5: Cattell & Horn's Model

Due to the difficulties in explaining data suggesting differential rates of decline in diverse cognitive domains with age subsequent researchers have sought to identify, describe, and explain the kinds of abilities compromised with age and those that are preserved. Cattell (1963; Horn and Cattell, 1967) suggested that there are two broad categories of cognitive ability subsumed hierarchically by g, or general intelligence. These abilities were called crystallised and fluid intelligence. Crystallised intelligence, according to Horn and Cattell (1967), represents an individual's accumulated knowledge, education, and expertise, for instance vocabulary and semantic knowledge. They suggested that crystallised abilities showed no age-related loss and in some cases improved with age. On the other hand fluid intelligence, reflecting abstract reasoning skills and the ability to acquire new knowledge, skills, and abilities, comprised those abilities subsumed by g that exhibit an age-related decline. Furthermore Horn & Cattell (1967), in common with many theorists, suggested that the decline of fluid abilities reflected the decline of the physiological bases of cognition: the brain, nervous system and the physical body and vital organs that support them. Clearly this theory has intuitive appeal since it describes a solution to the problem of differential decline in cognitive ability with age. Subsequent data have corroborated the idea that there are some abilities, reflected by fluid intelligence, that are likely to decrease with age while others, represented by crystallised abilities, showed no decline or even improvement. For instance, Schaie (1979) and Rabbitt (1993; Rabbitt et al. 2004) provided data suggesting that abilities described as fluid decline with age, but not appreciably until the mid-sixties. On the other hand both these large-scale longitudinal studies showed that abilities described as crystallised showed no decline.

3.6 Experience and Disuse Theories

One question this raises is whether or not this decline is inevitable. Consider Rabbitt's (1993) assertion that for some old people cognitive ability remains unimpaired in old age. Indeed some researchers have found that older participants can improve their fluid intelligence through practice and training (e.g. Willis and Schaie, 1986). This suggests that for many old people cognitive decline in fluid abilities may be preventable or avoidable, at least until one’s seventies. This clearly militates against any explanation that suggests that a broad set of fluid abilities will inevitably and monotonically decline with age.

Furthermore, it is uncertain that a decline in fluid intelligence has the same kind of impact on older people’s cognition that it has on younger people’s cognition. Stuart-Hamilton, Nayak, and Priest (2006), for example, followed work by Musch and Ehrenberg (2002) that had suggested that low fluid intelligence in younger participants may underlie an inability to reason probabilistically and a belief in the paranormal. Despite this, an equivalent relationship was not found for older participants, suggesting that fluid intelligence may not be as important in later life as in earlier life.

Another consideration is that expertise can ameliorate cognitive decline in well-practised areas, even in those tasks considered to reflect fluid abilities.

For instance Charness (1981, 1991) found that elderly expert chess players could compensate for their lack of fluid abilities through the use of well-practised strategies: using heuristics to reduce the processing load associated with developing novel solutions. Additionally Winder (unpublished, cited in Rabbitt, 1993, p. 398) found that experts' crossword solution ability was dissociated from fluid intelligence and that their ability showed a weak positive correlation with age ($r = 0.24$). In contrast both young and old novices' crossword solution abilities were positively associated with fluid intelligence and negatively associated with age. This implies that practice within a particular domain can more than compensate for any loss of fluid intelligence ability but that processes subsumed by fluid intelligence mediate performance in novel tasks. Furthermore Bosman (1993) found that younger typists had faster finger movements and reaction times than experienced older typists but could type no more quickly, although older but less experienced typists were more likely to make errors than any other group.

Taken together this evidence suggests that experience may have a vital part to play in attenuating age-related cognitive decline in specific, well-practised domains. Indeed Horn and Mazanuga (2000) suggest that one of the major weaknesses of the fluid intelligence theory of age-related cognitive decline is that IQ tests do not reflect expertise based on experience. They suggest that what is needed is a more complete taxonomy of cognitive ability: encapsulated in Horn's (1989) extended theory of fluid intelligence (see Horn et al. 2000 for a review). Having said that, the theory that age-related cognitive decline is best explained by a set of correlated abilities under the umbrella term of fluid intelligence and that this decline reflects, in turn, an underlying physiological decline still has its adherents. Certainly it makes sense to consider the possibility that when problems or tasks are completely novel that this may well be the case. Furthermore within the context of acquiring new stimulus-stimulus and stimulus-response associations a decline in fluid abilities may have more explanatory power than an expertise based explanation since, by definition, the ability to adapt to and act on novel information is considered a fluid ability.

Having said that, a note of caution is still justified when considering the impact of a decline in fluid intelligence and its relationship to disuse theories. One aspect that is difficult to quantify in this regard is older people's motivation towards complex cognition. Stuart-Hamilton and McDonald (2001) found that performance in concrete Piagetian tasks was related to age, fluid and crystallised intelligence, and Need for Cognition (Cacioppo, Petty, & Kao, 1984) scores. This suggests that more everyday reasoning is mediated by multiple factors, among them motivation for, and orientation toward cognitive effort as well as acquired knowledge. This means that fluid intelligence, whilst being a good predictor of performance in highly controlled laboratory tasks, may not be the only factor important in more everyday, concrete reasoning.

Another factor to consider in this regard, as well as for other reasons, is participants' level of education. Clearly it is a possibility that the greater the level of education the more likely participants are to be able to understand and complete experimental and psychometric tasks. Indeed it is certainly the case that simply controlling for years of education can attenuate the negative relationship between age and general fluid abilities (e.g. Ardila, Ostrosky-Solis, Rosselli, & Gomez, 2001; McCarthy, Sellers, Burns, Smith, Ivnik, & Malec, 2003; McCurry, Gibbons, Uomoto, Thomson, Graves, Edland, Bowen, McCormick, & Larson, 2001; see also Salthouse, 1991), and, for instance, list and name-face memory in older adults (Plude, Benaderet, Herrmann, 2001).

Several caveats must be born in mind, however. Firstly it is perhaps unsurprising that psychometric tests of fluid intelligence originally developed to predict educational achievement should be related to quantity of education. Secondly there is no possibility of knowing whether it is education *per se* that is instrumental in attenuating cognitive decline. It may well be, for instance, that education ‘primes’ people with practice at complex cognitive tasks and that although this ability may decline with age those with higher levels of educational attainment are still superior to less well educated members of their cohort in cognitive ability. It is equally possible, however, that a better education may lead to employment that is more cognitively demanding and that this promotes the use and development of complex cognitive abilities. Parallels can be drawn here to the observations alluded to in Chapter 2 concerning Easy-Hard transfer in discrimination learning and problem solving. It is possible that either education itself, or the consequent lifetime of cognitively demanding employment, may constitute the background learning that facilitates the solution of more abstruse problems or discriminations in the same way as Lawrence’s (1952) pigeons or Weisberg’s (1996) human problem solvers benefited from gaining experience from solving easy problems that led to an increased ability to address hard problems.

Another caveat to the education argument, however, is the point that higher levels of education are associated with higher socio-economic status and social support (e.g. Ross & Mirowsky, 1989; Rabbitt et al. 2004). This observation suggests that the elevated living standards of higher socio-economic groupings may attenuate the relationship between education and cognitive ability (c.f. Antonucci, 2001). On the other hand early education remains a good predictor of cognitive ability if socio-economic status is controlled for when lifelong level of intellectual activity is entered as a co-predictor (Kliegal, Zimprich, & Rott, 2004). Again, though, this suggests that cognitive preservation may be aided by more early education only if intellectual activity is sustained throughout the lifetime.

The previous paragraphs also relate to a further attempt to explain both age differences in cognitive ability and the observed increase in variability alluded to by Rabbitt (1993; Rabbitt et al. 2004). Clearly the suggestion that at least some age related decline may be attenuated by either expert knowledge and practice, or the long term results of higher levels of education leads one to speculate that simply using one's cognitive abilities may help to preserve them. Certainly it may be the case, as some theorists (e.g. Willis & Schaie, 1986) argue, that most older people's lives are fairly stable and that there is less need for what are considered fluid abilities, and instead a reliance on experience and knowledge. As a consequence cognitive skills that are little needed or practised decline because of lack of use rather than any inevitable age related decline. This view is consistent with disuse theory, more prosaically known as the 'use it or lose it' hypothesis.

Beyond the long term effects of education and practice there are some further strands of evidence that suggest the strength of a disuse explanation. For instance Pushkar, Arbuckle, Conway, Chaikelson, and Maag (1997) found that participant scores on the Everyday Activities Questionnaire (EAQ) accounted for some age related variance in cognitive ability. Similarly Shimamura, Berry, Mangels, Rusting, and Jurica (1995) found the cognitive abilities of university professors in their sixties comparable to those of university professors in their thirties. Likewise Schaie (1994) found favourable environmental conditions such as high income, cognitively complex employment lacking routine, activities such as reading, travel, club membership, and being married to someone of high cognitive ability attenuated age related cognitive decline. Again these observations suggest that keeping active and using one's cognitive abilities in hobbies and social and professional interactions may help preserve them.

Furthermore an enriched environment may lead to a quantitative increase in the number of synaptic connections per neuron in the brain. Hebb (1949), for instance, took an early and idiosyncratic approach to this question. He took some laboratory rats home and allowed them to roam his house and become pets for his children. Subsequently he found that these rats adapted more quickly and flexibly to new learning situations than the rats kept in his laboratory. This suggests that enriched environments may lead to an increase in 'plasticity', or cognitive flexibility: the ability to deal adaptively with new situations and challenges and to change one's thinking accordingly, a concept clearly allied to that of 'fluid intelligence'.

Subsequent studies have confirmed Hebb's (1949) hypothesis of use-induced plasticity. For instance Turner and Greenough (1985) found an increase in the number of synapses in rats housed in an enriched environment compared to caged rats, while Beaulieu and Colonnier (1988) procured similar results in cats. In humans histological studies such as Jacobs, Schall, and Scheibel (1993) have found an increased density of synaptic connections in Wernicke's area (a cortical language area) in those who had received a university level education.

Furthermore other researchers have found that brain plasticity is evident in ageing rats (Riege, 1971; Diamond, Johnson, Protti, Ott & Kajisa, 1985).

Consequently, the observed increase in experience contingent brain plasticity seems to be a robust finding that extends to several species, and continued use of cognitive ability may well attenuate or eradicate any age-related decline. Having said this it is equally possible that the apparent cognitive benefits of activity and use reflect the ability of the individual to carry out such activities. In other words activity may be constrained by, rather than facilitate, cognitive ability. The direction of causality is evidently unclear here, although it seems likely that continued activity on the part of those who are capable of such may be extremely beneficial. Certainly age related differences and individual differences in tasks deemed to require fluid intelligence are explicable by invoking the idea of experience induced plasticity, although it is unclear whether fluid abilities are determined by experience, or whether an individual's capacity to carry out activities is determined by existing or inherent cognitive ability.

3.7: Sensori-Motor Deficits

One further interpretation of the differences in results between fluid and crystallised intelligence tests concerns the observation that most of the former require time-limited tests whereas most of the latter have no time limits. For instance the AH4 and WAIS performance tests are both timed and are considered to reflect fluid intelligence (Salthouse, 1991; Horn et al. 2000). Most tests of crystallised intelligence concern vocabulary tests (e.g. MHV) and are untimed. This leads to the possibility that age differences in fluid intelligence are purely and simply due to an inability to perceive the questions and write down the answers as quickly as younger people.

To investigate this Storandt (1976) gave older people and younger people the digit symbol substitution task (DSST), a sub task of the WAIS involving the matching of digits to printed symbols according to a code (e.g. square = 2, triangle = 3, oblong = 4). Storandt found that much of the differences between age groups in DSST performance could be explained by a simple measure of how many symbols they could copy in 90 seconds. Furthermore Storandt (1977) found that some of the age-related variance in performance I.Q. was removed when time restrictions on the tests themselves were not imposed. Note, however, that by no means all age differences in performance are removed when sensori-motor speed is controlled for, or time limits removed. Thus, although sensori-motor speed may be a factor to consider when assessing the results of timed tests such as the AH4 this explanation is by no means able to account for all the observed age related differences in timed tests of fluid intelligence.

Other researchers consider the possibility that age differences in fluid intelligence may be due to performance deficits associated with poor eyesight and hearing. Granick, Kleban, and Weiss (1976) found a positive relationship between eyesight, hearing, and cognitive ability. In a more recent exhaustive review, Fozard and Gordon-Salant (2001) refer to relationships between both vision and hearing and cognitive performance. Furthermore visual and auditory acuity have been found to relate to a range of cognitive tasks in a six-year longitudinal comparison among 418 older adults (Valentijn, van Boxtel, van Hooren, Bosma, Beckers, Ponds, & Jolles, 2005). Certainly it is possible that poor sensory acuity may adversely affect participants' ability to apprehend and follow instructions, whether written or verbal. One question they raise, however, is that any relationship between hearing or vision and cognition may be due to perceptual processes rather than sensory acuity *per se*. Poor visual or auditory acuity may, for instance, lead to problems in distinguishing between stimuli and a consequent deficit in observed cognitive ability.

3.8: Health Status

Another factor that may be causal with regard to age related cognitive decline is that cognitive decline reflects a parallel decline in health. It seems reasonable to argue that any physical illness may well have an impact on the brain and central nervous system, and consequently upon cognition. Indeed Jelicic and Kempen (1999) found that poor self-rated health adversely affected cognitive performance. Although one could argue that self-rated health is a poor measure of general, overall health there appears to be a robust relationship between this measure and medical ratings of general health (e.g. Maddox and Douglas, 1973); and between self rated health and diagnosed medical conditions (e.g. Pilpel, Carmel, and Galinsky, 1988).

Despite this the relationship between general health and cognition remains unclear since studies screening participants for medical problems show the same patterns of age related decline in cognition as those that do not (e.g. Botwinick & Birren, 1963; Albert, Wolfe, & LaFleche, 1990), implying that poor general health, although related, may not be a reliable predictor of cognitive decline in old age. Furthermore ill health has been associated with basic perceptual speed measures but not higher order processes such as attention and episodic memory (Rosnick, Small, Graves, & Mortimer, 2004), suggesting that health status may differentially affect a range of cognitive abilities.

There is, however, an abundance of evidence to suggest that specific serious illness may have a profoundly negative impact on cognitive ability. For example Zelinski, Crimmins, Reynolds, and Seeman (1998) found that diabetes, high blood pressure or stroke had a negative impact on basic cognitive abilities. Van Boxtel, Buntinx, Houx, Metsemakers, Knottnerus, and Jolles (1998) found cognitive decline in older people exacerbated by heart disease, circulatory disorder, and bronchitis or other respiratory disorder while Streisand, Rodrigue, and Sears (1999) found evidence relating liver and kidney disease to cognitive decline. Finally Ebert and Heckerling (1998) related cancer and Parkinson's disease with cognitive deficits, as well as highlighting glaucoma or cataracts' negative impact on participants' ability to communicate and understand instructions: important in an experimental context.

More recently, however, Hebert, Scherr, Bennett, Bienias, Wilson, Morris, and Evans (2004) found that blood pressure was not a significant predictor of cognitive ability in later life in a sample of 4284 individuals living in communities where medical treatment of blood pressure was common. This raises the possibility that medical intervention may ameliorate the effects of poor health on cognitive decline. Furthermore Barusch, Rogers, & Abu-Bader (1999) found a relationship between depression and cognitive decline, and in a more recent study anxiety and depression were found to be related to cognitive decline over six years whereas general physical health was not (van Hooren, Valentijn, Bosma, Ponds, van Boxtel, & Jolles, 2005).

This, again, demonstrates the ambiguity of the relationship between health and cognitive decline. It is unclear whether the two are intimately related or clearly separable, although it makes more sense to assume the former since cognition relies upon the brain, which is merely another part of the physical body and just as subject to decay over time.

Indeed some researchers (e.g. Kleemeir, 1962; Riegal & Riegal, 1972) suggested that a sudden loss of cognitive ability was a predictor of imminent death. This idea was formalised as the somewhat pessimistic 'terminal decline' theory that explains individual variability in cognitive ability as reflecting the impending failure of the body. There are, however, problems with this conception since there is no agreement on which particular skills predicted the approach of death, and furthermore this effect seems to be restricted to the under seventies (White & Cunningham, 1988). More recent evidence, on the other hand, suggests that cognitive decline is a good predictor of an increased risk of hospitalisation (Chodosh, Seeman, Keeler, Sewall, Hirsch, Guralnik, & Reuben, 2004).

If this is the case then it may lead to a reassessment of the terminal drop hypothesis, and it may be that decline does not predict death but can forewarn of impending illness serious enough to require inpatient attention. This conclusion is in accord with Bosworth and Sieglar's (2002) meta-analysis of terminal decline studies: rapid cognitive decline is more likely to be associated with physical decline and the aetiology of Alzheimer's disease than with impending death.

Another possibility is that since illness almost inevitably means more medication then it may be that prescription drugs, rather than illness as such, may cause cognitive decline. A recent study found that neuroactive drugs and multiple drugs were associated with cognitive decline, although Statins, used to treat cardiovascular disorders, seemed to ameliorate the adverse effects of age in this respect (Starr, McGurn, Whiteman, Pattie, Whalley, & Deary, 2004). The latter effect is, however, more often associated with the prevention or delay of the symptoms of dementia. Specifically Statins' role in reducing cholesterol may help prevent the cardiovascular problems and amyloid plaques associated with a predisposition to Alzheimer's disease and vascular dementia (Naidu, Xu, Catalano, & Cordell, 2002).

Whilst differential physiological decline for exogenous reasons may be a reasonable explanation of individual variation in cognitive ability in the later years one cannot ignore the alternative, or complementary, explanation that cognitive decline with age may be equally well explained by endogenous factors. For instance McClearn and Vogler (2001, McClearn, 2002) review evidence to suggest that the rate of physical and cognitive decline with age may be linked to genetic factors. Other reports suggest that genetic variations may dictate the rate of cognitive decline and risk of dementia, cardiovascular function and disease, vulnerability to stress, longevity, memory, and intelligence (Deary, Wright, Harris, Whalley, & Starr, 2004). Most interest in this area has been directed at the epsilon 4 allelle of the apolipoprotein 4 gene, and a meta-analysis of 38 studies revealed the epsilon 4 allelle was associated with declines in general cognitive ability, episodic memory, and executive ability (Small, Rosnick, Fratiglioni, & Backman, 2004).

However even they would not suggest that one can explain all physiologically linked age-related cognitive decline purely through genetic factors, and a recent study reported that epsilon 4 allelle was not associated with AH4 test scores in a sample of 767 older adults (Rabbitt et al. 2004). Certainly it is unlikely that individual differences in rate and extent of decline are entirely the result of our genes, particularly since it is probable that having lived longer almost undoubtedly leads to greater exposure to and influence from the environment. Even this seemingly simple assumption, however, may ultimately be proved wrong since recent estimates put the level of genetic influence on cognitive ability in later life at over 50% (Plomin & Spinath, 2002). Of course it is notoriously difficult to separate genetic from environmental influences and the means to do so are beyond most researchers. Hence the task of most psychological research has been to determine the nature and extent of the interaction between ageing and the environment.

3.9: The Age by Complexity Effect

One further consideration with regard to age-related declines in cognitive tasks is the age by complexity effect. Put simply this concerns the common observation that a systematic increase in task complexity brings about a comparable increase in any observed age-related deficits. For instance Bottwinick, Robbin, and Brinley (1960) found that, in a card sorting task age differences increased systematically with the number of sets participants were required to sort the cards into. Furthermore Salthouse, Mitchell, Skovronek, and Babcock (1989) gave verbal reasoning and paper folding tasks requiring visualisation and transformation that varied in terms of the number of premises or folds required to complete the task. They found statistically significant correlations between age and task complexity of 0.43 and 0.45 respectively, demonstrating that as task complexity grows so too do age-related differences.

This is just one of a plethora of studies that have demonstrated an age by complexity effect (e.g. Clay, 1954; Welford, 1958; Kinsbourne, 1980; Wingfield, Poon, Lombardi & Lowe, 1985; Klatsky, 1988) and it seems to be a robust empirical phenomenon. There are, however, several caveats to this view. Firstly what is termed complexity is a relatively vague concept and there is no clearly agreed way of operationalising this variable. Secondly, as Salthouse (1991) points out, researchers have tended to emphasise data that support the predictions of the age by complexity theory and ignore those that refute it. There is certainly a case for more research that systematically varies complexity.

3.10: Brinley Plots

One way of looking at systematic variations in task complexity and their effects on performance in cognitive ageing studies has been to use what are termed Brinley plots, named after their originator, James Brinley. Brinley (1965) took mean error scores from a range of reaction time studies of varying complexity and plotted old and young scores against each other in a scatterplot, with older participants' scores on the abscissa and young on the asymptote. He found that the resulting figure showed a linear relationship between the task performances of the two age groups that could be described using a regression equation. Furthermore, the slope of the line was greater than 1 and had a negative intercept with the abscissa, suggesting that the performance deficit of older participants increased systematically with task complexity and that the performance deficit was caused by a general underlying factor. This observation lay dormant for a number of years, principally because Brinley (1965) made few theoretical claims for the relationship. Cerella (1985) used the technique in a meta-analysis of 35 studies of response latencies, finding that two factors were necessary to predict older participants' performance from younger participants' performance: a general sensori-motor, or peripheral slowing factor, and an estimate of the extent that central processes were involved in the task. Cerella (1985) concluded that a large proportion of the age differences in reaction time were probably due to a general slowing factor. Other researchers took the concept still further. Salthouse (1991, pps. 313-317) cites such studies in support of the notion that age differences in cognitive abilities are due to a reduction in processing resources, since observed age differences increase systematically as resource requirements increase. Indeed, such observations laid some, but by no means all, of the foundations for Salthouse's (1996) processing speed theory of cognitive ageing, discussed in the next chapter. Whilst it seems compelling that one can predict older from younger people's task performance in a range of tasks with reasonable accuracy using a simple linear regression model there are real concerns with the interpretation of Brinley plots. Perfect (1994; Perfect & Maylor, 2000) reminds us of that simple truism; correlation does not imply causation, in his questioning of the method. Simply because task performance is predictable in this way does not mean that task performance in all of the studies analysed by, for instance, Cerella (1985) was caused by the same factor. Indeed, Cerella (1985) found that better predictions were derived from treating each study individually, but put this down to a variation in the proportion of central and peripheral processes engaged in the different reaction time tasks. Perfect and Maylor (2000) suggest that the use of group means inflates the predictive power of the regression equations, citing an example (Fienberg, 1971, cited in Perfect & Maylor, 2000, p.13) that the probability of being selected for the US draft decreased the later one's birth date was, but that analysis by birth month increased this correlation from *rho* = -0.226 to a staggering *rho* = -0.839. This demonstrates a huge difference between individual and group mean analysis and reinforces the point that such analyses may overestimate the extent to which age differences can be predicted by a general factor. Even if one assumes the Brinley plot analyses are valid in this sense, there are other criticisms of the methodology. Ratcliff, Spieler, and McKoon (2000; Ratcliff, Thapar, & McKoon, 2001; Ratcliff, Spieler, & McKoon, 2004) are equally vocal in their criticism of Brinley plots. Their argument rests on the assertion that Brinley plots are what is termed by statisticians quantile-quantile (Q-Q) plots. Q-Q plots are constructed by rank ordering the scores arbitrarily on each axis, and are generally held to reflect the ratio of standard deviations in each population. Thus, Ratcliff et al.'s argument is that all that the slopes of Brinley plots tell us is that variability in performance increases with age, an uncontroversial and common observation that tells us little of what causes age related cognitive decline. Furthermore, they argue that much of the observed age differences in reaction times can be attributable to the fact that older people are more cautious in tasks and emphasise accuracy over speed. Myerson, Adams, Hale, and Jenkins (2003), however, argue that Brinley plots are not Q-Q plots since the data are unranked: the appearance of ranking is an artefact of systematic task differences, and that older people are not more cautious in their approach to reaction time tasks. This, however, is inconsistent with results reported by Ratcliff et al. (2001, Experiment 2). In a study where participants had to make decisions about whether distances between dots displayed on a computer screen were 'short' or 'long' old and young groups were given alternating instructions emphasising speed or accuracy. Ratcliff et al. (2001) found that both types of instruction produced typical Brinley plots when mean old and young response latencies for progressively more difficult discriminations were plotted against each other, but that the slope of the Brinley plot for accuracy was greater than that for speed instructions. If one then plots the speed condition data for older participants against accuracy data for younger participants, however, the slope becomes less than 1, implying that older people are faster than younger people at responding to stimuli and that their general cognitive processes were faster, not slower. Ratcliff et al (2000, 2001, 2004) have argued that there is no way of telling whether participants tend towards speed or accuracy in any experiment, and it is possible that all Brinley plots that plot old and young response latencies are telling us is that older people are more likely to emphasise accuracy than speed. Certainly, a lifetime's experience of the inadvisability of hasty decisions as opposed to the stereotypical impetuousness of youth makes this interpretation intuitively plausible. Furthermore, Cerella (1985) explicitly stated that the Brinley plots he analysed emphasised speed rather than accuracy, and were thus only half the story, and concurred with Perfect and Maylor's (2000) assertion that analyses based on group data would not necessarily hold true for individuals. Overall, Brinley plots tell an interesting story about the consistency of age related declines in reaction times and point to the possibility of a single or limited range of factors that may determine cognitive decline with age, but are flawed as a means of analysis.

3.11: Conclusions and Directions

Of course, one may recognise the observation that age-related declines in most cognitive tasks are related to, and predicted by a co-occurring decline in fluid intelligence. In addition one needs to explain why more complex tasks exacerbate age-related differences in a variety of cognitive tasks. Furthermore invoking a 'fluid intelligence' explanation cannot explain Rabbitt's (1993; Rabbitt et al. 2004) observations concerning the individual differences in rate and extent of decline for fluid intelligence tasks. This obviously begs another question: why should fluid intelligence decline with and interact with task complexity and individual differences? This question can broadly be addressed with disuse theories and explanations based on greater prevalence of somatic illness or sensori-motor deficits. This is, however, insufficient since these theories simply predict an overall age-related deficit. In order to generate novel predictions concerning associative learning it is necessary to look more deeply into specific age-related cognitive and neuropsychological deficits. Consequently the following chapter will examine specific and general process deficits that have been postulated to underlie cognitive decline, as well as look at neuropsychological evidence for cognitive decline that can be related to the physiological bases of learning discussed in Chapter 2. This will make it possible to integrate what is known about associative learning with knowledge concerning age related cognitive decline and suggest predictions concerning the nature of any putative decline in associative learning abilities with age.

Chapter 4: Theories of Cognitive Ageing

4.1 Introduction

So far we have established that age-related cognitive decline applies neither to every individual nor to all cognitive domains. Instead variability in cognitive ability seems to increase with age, seems to be confined to a set of cognitive abilities related to working flexibly with new information known as fluid intelligence, and that the extent of any decline is exacerbated by task complexity. We have also looked into general factors that may underlie individual differences in age-related cognitive decline, such as health, fitness, expertise, disuse, reduced brain plasticity, methodological confounds, and sensori-motor deficits. While these factors may be of interest in their own right in the context of the present study it is desirable to take into account more detailed, largely process specific theories of cognitive ageing. This is important since theories of associative learning are postulated in terms of cognitive and neuropsychological resources.

There are several theories of cognitive ageing that are relevant to learning and memory, and therefore to the present investigation. One theory of memory decline with age has already been described: the suggestion that memory declines monotonically with an underlying fluid intelligence factor. As we have already seen this may be a less than convincing explanation for that decline, since fluid intelligence may reflect many basic processes requiring multiple resources. Other theories have been developed to account for memory changes in later life. These include the processing speed theory of cognitive ageing (Salthouse, 1996); inhibitory failure (Hasher & Zacks, 1988); source monitoring failures (Johnson, Hashtroudi, and Lindsay, 1993); the frontal lobe theory (West, 1996); and the associative deficit hypothesis (Naveh-Benjamin, 2000; Naveh-Benjamin, Craik, & Ben-Shaul, 2002; Naveh-Benjamin, Hussain, Guez and Bar-On, 2003; Naveh-Benjamin, Guez, Kilb & Reedy, 2004). There are, in addition, a great many data describing neuropsychological changes with age that may be directly relevant to the theories of learning discussed in chapter two.

4.2 The Processing Speed Theory

Salthouse (1996) proposed a theory suggesting that broad age-related declines in fluid cognitive abilities were related to a systematic decline in the speed at which older people process information. Salthouse's (1996) theory hinges on extensive empirical observations in multiple cognitive domains related to fluid intelligence and mnemonic abilities (e.g. Salthouse, 1991; 1992; 1993; 1994; 2000 Salthouse & Babcock, 1991). Salthouse observed that generalised age related changes in cognitive ability could be minimised or even eliminated by statistical control of measures of processing speed. Furthermore he proposed that speed of processing mediated cognitive ability through two separable mechanisms: the limited time mechanism and the simultaneity mechanism.

Fundamentally the limited time mechanism means that slowed processing of information leads to a cumulative decrement in performance, since each process takes longer to complete. This effectively means that older people will perform subsequent cognitive operations later than younger people, and that this difference will increase as time progresses causing operations to be processed too slowly to be completed. This, as it stands, may be a reasonable explanation of age differences in simple serial tasks. The cumulative effect of slowed processing on more complex tasks contingent on multiple sub-tasks creates a further disparity between old and young, since each sub-component of a complex cognitive task must be completed within a limited time.

Salthouse's proposed simultaneity mechanism concerns the limitations associated with time based loss of the products of earlier processing in complex tasks through decay or displacement. The assumption here is that information deteriorates over time and the products of earlier processes available for later processing are degraded or even lost if ensuing cognitive operations are too slow. Put more simply Salthouse suggests that much of the observed age related decline in cognitive ability is caused by slower processing of information leading to fewer operations per time period and a compromised ability to maintain and use the products of previous processes. This conception is not an unreasonable one, and can also account for empirical observations such as the age by complexity effect and decrements in time based tests.

Salthouse's (1996; 2000) evidence is based on the macro approach introduced in the previous chapter. The processing speed theory assumes that cognitive ageing is characterised by a general and monotonic decline in ability, and is therefore a natural successor to the psychometric approaches that explained cognitive ageing by invoking a general or fluid intelligence explanation. Furthermore, it assumes that declines in various cognitive domains are not independent, but are related to and therefore explicable in terms of a general factor, in this instance processing speed. Salthouse (1996) presents a plethora of evidence to support the hypotheses that firstly there is a general 'speed' factor that predicts and mediates much of the relationship between age and most specific cognitive abilities, and secondly that this mediation can be largely explained through limited time and simultaneity mechanisms.

For instance Salthouse and Coon (1993) found that relationships between age and arithmetic ability were rendered non-significant after controlling for perceptual speed and reaction time. Similarly Salthouse and Meinz (1995; Salthouse, 1996) found that comparable controls mediated age differences in a Stroop (1935) colour-word task. Equivalent results have been found for long term memory for activities (Earles & Coon, 1994), paired associate learning and free recall (Salthouse, 1993), working memory (Salthouse & Babcock, 1991) and many other cognitive tasks (see Salthouse, 1991; 1996; 2000; Madden, 2001 for reviews). The relationships between measures of perceptual speed and reaction times with age related cognitive decline seem to be a robust phenomenon, and Salthouse's claim that this underlies much observed age related cognitive decline still attracts support from many corners as a parsimonious and tractable theory.

The specific mechanisms Salthouse suggests have some empirical support too. Salthouse (1996, p. 422-423) cites unpublished evidence from Kersten and Salthouse (1993) in support of the limited time mechanism. Participants were given a continuous associative memory task requiring them to view letter-digit pairs, and then tested by deciding whether subsequent probe pairs had been presented together. Probe pairs were presented either immediately afterwards, or after presentation of another letter-digit pair. Results indicated that accuracy improved as a function of presentation time for all stimuli in a classic negatively accelerated 'learning curve'. Participants overall performed worse on trials where alternative pairs were presented between stimulus exposure and recall, and this is interpreted as demonstrating that greater time was needed for processing in this condition. Younger participants' performance in this condition was also superior to older adults' performance in the immediate recall condition, and Salthouse infers that this suggests older adults complete less processing per time unit than younger people. Although this interpretation seems reasonable all these data really tell us is that older people perform far worse when recognising previously seen letter-digit pairs and that although they are worse at this task when alternative pairs are placed between stimuli and probe they suffer no worse a decrement than younger people. Furthermore, although this suggests the kind of monotonic decline with age associated with general factor theories of ageing it doesn't necessarily follow that this general factor has to be processing speed *per se* since it is equally possible that older people may have had difficulty learning the letter-digit associations, or that their memory for them has degraded.

Similarly, Salthouse cites evidence in support of the simultaneity mechanism by comparing this to the concept of working memory. It is suggested that the two are synonymous since both concern the amount of information that can be 'worked on' at any one time. Salthouse (1994; see also Craik, 1986; Light, 1991) reviewed studies of working memory and age and found, unsurprisingly, age related decline. Importantly for his theory these declines were largely attenuated by statistical control of processing speed parameters where these measures were available. For instance Salthouse and Babcock (1991) tested 233 older adults on a composite reading and computation span task and found that age explained 21% of the variance in these measures. Once perceptual speed was accounted for, however, age only accounted for 0.7% of the variance. Again although this account seems compelling it merely shows an association between measures of working memory and measures of what Salthouse calls 'perceptual speed'. Suggesting this shows that processing speed causes working memory decline with age, or that working memory is analogous to the putative simultaneity mechanism underlying a general speed related deficit isn't necessarily the only interpretation available. The concomitant decline in reaction time, perceptual speed and general measures of cognitive decline with age may be coincidental; hence the attenuation of age deficits by measures of speed of processing. Equally, it may be that working memory deficits constrain processing speed. Whatever the interpretation all these data show us is that working memory abilities and processing speed decline with age, which merely confirms what Perfect and Maylor (2000) call 'the dull hypothesis'; that cognitive abilities decline with age.

Other criticisms of the processing speed hypothesis suggest that the measures of perceptual speed may not be as basic as Salthouse claims. For instance Parkin and Java (1999; 2000) suggest that the Digit Symbol Substitution Task (DSST; Wechsler, 1958) requires more than simple perceptual processes for successful completion. Since this task is suggested by Salthouse and other proponents of the processing speed theory to reflect perceptual speed it should be so simple that it does not engage higher cognitive abilities. Parkin and Java (1999; 2000) pointed out several reasons why this may not be the case. They suggest that since the DSST is a performance subtask of the WAIS R (Wechsler, 1987) and correlated with other measures in this battery it is entirely understandable that it should attenuate age related cognitive decline in a broad range of 'fluid' tasks. Further they analyse the assumptions of the DSST itself, which involves a code table with the digits one to nine placed above a corresponding row of symbols. The task requires participants to place as many of the appropriate symbols in boxes below a series of random single figure digits from one to nine as they can in 90 seconds. Parkin and Java suggest that this task has a substantial memory component since participants are required to learn associations between digits and symbols and maintain them in memory as they complete the task.

Furthermore Parkin and Java (1999; 2000) carried out a series of regression analyses demonstrating that DSST scores predicted performance on the AH4 group test of general intelligence and vice-versa. Additionally DSST and AH4 scores also predicted performance in other recognition and recall memory tasks. Entering DSST scores first in a hierarchical regression analysis resulted in the AH4 predicting little of the variance beyond this. Likewise entering AH4 scores first in the analysis attenuated the DSST's power to predict memory ability, although not to the extent that DSST scores attenuate the predictive value of AH4 scores.

As pointed out earlier, it should be no surprise that a sub task from a general test of intelligence should predict performance on another test of general intellectual ability. Recall, too, that Storandt (1976) found that much of the age difference in DSST performance could be explained by the number of symbols participants could copy in a specified time, suggesting that the task may involve a considerable sensori-motor component. It is therefore possible that the processing speed theory is predicated on the basis of a combination of general intellectual ability and sensori-motor, rather than perceptual, speed. Although this criticism is valid for the DSST it is only fair to point out that Salthouse's theory is not solely based on DSST performance, but includes evidence from many much simpler measures of perceptual speed such as the digit copying task. This simply requires participants to copy as many digits as they can in a specified time, although this may also involve a considerable sensori-motor component, and similarly the letter and pattern comparison tasks simply ask participants to state whether two letters or patterns are the same or different. Again the dependent variable here is how many items correct participants score in a specified time, usually 30 seconds. Other researchers (e.g. Zimprich, 2002) have solely used the DSST as an index of perceptual speed and the results of such studies should be viewed with some caution. The matter of how to quantify perceptual speed does, however, raise a serious question: just what *is* it?

Salthouse himself admits that this is a difficult question to answer, although he contends that because the various measures of perceptual speed that he uses are highly correlated and able to predict performance on a number of other cognitive tasks that the theory has some validity. In addition at least one recent study (Walhovd, Fjell, Reinvang, Lundervold, Fischl, Salat, Quinn, Makris, & Dale, 2005) has used physiological Event Related Potential (ERP) latencies as a measure of processing speed in conjunction with MRI estimates of cortical volume. This study found that ERP latencies increased with age and was a significant predictor of WAIS performance, suggesting that Salthouse's theory may have some physiological basis. The same study, however, also found that cortical volumes decreased with age and were a better predictor of WAIS scores. Additionally Walhovd et al. (2005) found that ERP latencies and cortical volume were not significantly related in the final analysis, suggesting that although processing speed may predict some age related cognitive decline cortical atrophy may play a bigger and complementary part. Furthermore many misinterpret Salthouse's theory as suggesting that processing speed, however measured, is a fundamental cause of all age related cognitive decline. Salthouse himself is far more reticent, and goes to great lengths to make it clear that processing speed is a major, but not by any means the only determinant of cognitive decline. One further criticism is, however, that it is unclear precisely what causes reductions in processing speed with age, particularly in the light of the observation that processing speed seems dissociated from cortical volume (Walhovd et al. 2005).

It is fairly simple to suggest what processing speed theory might predict for associative learning tasks. Since the theory predicts a monotonic decline for all cognitive abilities mediated by task complexity this is what one should find, in addition to the prediction that much of the age related variance should be attenuated by controlling for perceptual speed. Therefore PPPs and NPPs should be equally challenging, and there should be no differences between solving a biconditional problem AB+, CD+, AD-, BC- and a conditional problem EG+, EF+, HG-, HF- since the loads for processing stimulus-stimulus and stimulus-response associations are equally demanding.

Note, however, that one could suggest that nonlinear problems are more demanding and therefore ‘complex’ than linear ones, and consequently subject to age related decline. This leads to another problem for the processing speed theory. Although Salthouse (1996; 2000) suggests that ‘more demanding’ or ‘complex’ processing leads to a greater age related cognitive deficit there is little in the theory that makes firm predictions about these observations or explicitly states exactly what constitutes complexity. Furthermore, evidence suggests that memory processes may be dissociable and show differential decline. Salthouse dismisses these findings as reflecting an invalid ‘micro’ approach, instead emphasising the general nature of cognitive decline for cognitive abilities he suggests are based on fluid intelligence. Although it is certainly more succinct to suggest this it does mean that details are lost in the quest for parsimony. The following sections review the evidence on ageing and memory, and some of the 'micro' approaches that have sought to explain them.

4.3: Ageing and Memory

One of the major findings concerning age related deficits in memory is that conscious, episodic memories are more greatly affected than implicit, unconscious and semantic ones and that encoding and retrieval processes are disproportionately subject to the effects of ageing relative to storage (Cohen, 1986; Light, 1991; Wechsler, 1997; Backman, Small & Wahlin, 2001; Ronnlund, Nyberg, Backman & Nilsson, 2005), suggesting that operations requiring effortful processing are disproportionately compromised with age. This is certainly consistent with the view of fluid decline with age, since fluid processes by definition require conscious, effortful processing. It is also consistent with the greater decline older people suffer in terms of free-recall memory relative to cued-recall which is in turn poorly preserved compared to recognition memory.

Semantic memory is generally spared with age, unsurprising since this is undoubtedly involved in crystallised intelligence as assessed by such instruments as the MHV. This suggests that older adults tend to have little problem with isolated pieces of knowledge and is consistent with the suggestion that storage is relatively preserved in age. Similarly procedural memories and the acquisition of motor skills usually show no age related decline (Light & LaVoie, 1993), and neither does implicit memory as measured by repetition priming tasks such as word fragment completion or fact completion (Backman et al. 2001).

Prime among candidates for memory decline, as Salthouse observed, is working memory and the attentional and inhibitory processes associated with it.

Other areas of interest in the ageing and memory literature are prospective memory, source memory, and associative memory.

4.4 Working Memory

Working Memory (WM) is a powerful and widely accepted explanation for short term, or immediate memory (STM). Baddeley and Hitch (1974; Baddeley, 1986) first suggested that STM was more than just a passive store that memories passed through to get to long term memory (LTM), as Atkinson and Shiffrin (1968) had initially proposed. Instead they promulgated the notion that WM involved effortful, deliberate processing that required conscious attention and the control and management of limited resources, such as attention and capacity. The major function of the WM system is postulated to be the allocation of these limited resources to salient stimuli while inhibiting their allotment to non-salient stimuli, thus ensuring efficient processing (e.g. Miyake & Shah, 1999).

A prototypical measure of WM illustrates this point. In Daneman and Carpenter's (1980) listening span task participants are required not only to determine the truth or falsehood of a sentence, but also to remember the last word of each. Clearly this task and others like it require two different processes to be executed in parallel, verifying the sentence's truth and remembering the final word in each block of sentences. Again Salthouse's (1991, 1994, 1996, 2000; Salthouse & Babcock, 1991) extensive observations give us data strongly suggesting that WM declines with age. Since the capacity of short term memory, as assessed by digit span and similar measures (see Backman et al. 2001), shows only small declines, if any, it seems reasonable to suggest that other, more complex processes mediate age-related WM decline. Salthouse's interpretation is that WM declines are due to reduced processing speed. Indeed he suggests that his simultaneity mechanism is synonymous with working memory and that this works in parallel with a limited time mechanism to produce age related cognitive decline.

Others point to attentional deficits especially when participants are asked to switch focus during a WM task and in divided attention tasks related to higher order executive abilities rather than basic perceptual speed (Verhaeghen & Cerella, 2002; Verhaeghen & Basak, 2005). Furthermore it is equally compelling to argue that reductions in processing speed are due to WM limitations, so Salthouse's admirably comprehensive analysis may border on the tautological in this instance. Other researchers have, however, found comparable declines in WM with age and similar relationships with measures of processing speed (de Ribaupierre, 2002; Backman et al. 2001; Chaytor & Schmitter-Edgecombe, 2004; Waters & Caplan, 2005). Doubtless WM does decrease with age, but it is a broad concept, and to generate predictions concerning associative learning it would be better to look at the specific processes underlying WM.

4.5 Inhibitory Processes

Hasher and Zacks (1988; Zacks & Hasher, 1994) proposed that an inhibitory deficit may be responsible for age related differences in WM tasks and episodic memory in particular, and also for cognitive decline in general. They suggest that older people's inability to actively suppress, or inhibit, irrelevant information means that WM capacity is wasted on unnecessary items and renders older people more prone to interference, causing the observed pattern of age related deficits in fluid intelligence.

There certainly is evidence that inhibitory processes are compromised with age. Hasher, Stoltzfus, Zacks, and Rypma (1991), for instance, found that younger people inhibited responses to irrelevant stimuli in a letter naming task more than older participants. Here letter targets were indicated by colour, and half of trial blocks were sequential in that the previous trial's irrelevant letter served as the following trial's target whereas control blocks were made up of random two letter pairings. Older adults made more errors and took longer to react to stimuli overall, and equally long for both trial types. Younger adults were quicker overall and took significantly longer to respond to sequential than to control trials. This implies a degree of conscious contemplation in sequential trials not present in control trials. Furthermore younger people were far more likely to notice the pattern in sequential trial blocks, indicating they were better able to distinguish between trial types and to make predictions based on consciously known environmental consistencies. This is analogous to the rule learning effect found by Shanks and Darby (1998), and does nothing to disconfirm their hypothesis that learning of initial contingencies is a necessary precursor of rule induction. The results described above suggest that younger people seem to allocate more effortful processing and conscious resources in inhibiting irrelevant stimuli, and that their superior performance allows them to induce rules to help them predict outcomes.

In a similar task Kane, Hasher, and Stoltzfus (1994), using familiar words (e.g. cat, pot) instead of letters, demonstrated that older adults were more likely to process irrelevant stimuli and were less able to suppress their later activation. Mani, Bedwell and Miller (2005) found that age differences were greater for false alarms in a continuous performance task than for omission errors, suggesting an inability to inhibit inappropriate responses.

Hedden and Park (2001) used manipulations of an AB-CD retroactive interference task to explore inhibitory processes. In this task paired associates (e.g. door, lock; AB) are learned in stage 1, followed by either a rest condition or further read paired associates (e.g. region, place; CD) and a recall stage where participants were asked to say whether test pairs had appeared in stage 1 or not. Stimuli were presented on a computer monitor and duration of both presentation and read or rest stages held constant for all participants. They found older adults were more prone to errors and false alarms than younger participants in the read as opposed to the control condition at test, as well as slower to respond. Reaction time in this task did not mediate the increased retroactive interference found with age, suggesting that simple reaction time may not be a good predictor of resistance to interference in this instance. Rather, this suggests that resistance to interference is associated with higher cognitive functions, and is consistent with inhibitory failure since here older people are unable to suppress activation of paired associates learned in stage 2 when asked to ignore these and simply identify stage 1 word-pairs.

Certainly this is applicable to associative learning, since this could lead to an inability to differentiate between similar stimuli, and whether a stimulus led to a response or not. Recall that Shanks, Charles, Darby, and Azmi (1998) and Shanks, Darby, and Charles (1998) found resistance to retroactive interference in a contingency task beyond that predicted by either elemental or configural models of associative learning. This implies, in younger people at least, a greater ability to inhibit inappropriate responses to stimuli resulting from generalisation of perceptual similarity than Pearce's (1987, 1994, & 2002) model suggests. This generates the prediction that effortful inhibitory processes can be engaged by younger people to facilitate retention of learned discriminations. Clearly, therefore, older participants should suffer greater retroactive interference, or the disruption of old learning by new learning, than younger participants. Given the evidence discussed above this seems a more than reasonable prediction.

Furthermore, studies have demonstrated that older participants are also more prone to proactive interference in working memory tasks as well. Bowles and Salthouse (2003), for instance, used computation and reading span tasks to look for evidence of proactive interference. They found that later trials were more difficult for older participants than younger participants, and that the differences in performance attributable to this proactive interference accounted for about half of the age related differences in performance for both working memory tasks. Again, this suggests a vulnerability to interference as a result of an inability to suppress activation of redundant memories.

Similarly, Andres, Van der Linden, and Parmentier (2004) used a directed forgetting paradigm to investigate age differences in interference in working memory. Participants had to remember letter trigrams in a single trigram condition, an interference condition where two trigrams were presented and both had to be remembered, and a directed forgetting condition where they received instructions that the second trigram was to be forgotten. There were no age differences in the single trigram condition; presumably this was low in terms of complexity and working memory load. In the interference condition both young and old participants performed worse, but older people's performance decline was greater, suggesting they are more prone to interference in memory. In this condition errors were characterised by omission of letters from the trigrams rather than intrusion or contamination from other stimuli, or errors in the position of letters within the trigrams. A similar pattern of results emerged in the directed forgetting condition in that older participants made significantly more errors and there was a significant difference in terms of omission errors. There were, however, significant age differences in terms of intrusion errors in that older participants were more likely to recall letters from trigrams that they had been directed to forget. This demonstrates a further susceptibility to retroactive interference since the later to be forgotten trigrams were confused with the earlier to be remembered trigrams, but provides an explicit explanation for this observation in that older participants were less able to inhibit irrelevant information in working memory. Despite this, an inhibitory explanation of the interference condition is difficult, since participants are not specifically directed to ignore any information although this does not rule out the possibility that inhibitory processes were necessary for successful task completion. The authors suggest that the omission errors seen in both the interference and directed forgetting conditions could simply be due to diminished capacity in working memory. Although age differences are not generally apparent in simple span tasks for short-term memory (e.g. Salthouse, 1991; Backman et al. 2001) it is possible that a reduction in processing speed, and in particular deficits associated with a simultaneity mechanism, could explain these observations. In any event it does seem plausible that inhibitory processes weaken with age and may lead to increased errors and overgeneralisation in associative learning tasks, especially when similar stimuli lead to different outcomes and when stimuli are re-valued in different stages of an experiment.

Hedden and Park (2001, 2003), however, suggested that interference related deficits in WM with age might be equally well explained by source memory deficits, that is, an inability to remember the context in which memories were formed, or where a specific piece of information was learned. This explanation could be equally compelling in explaining the above results: intrusion errors could have occurred simply because older participants could not remember the context in which they learned the particular elements within the trigrams

4.6 Source Monitoring Deficits

Source monitoring can be defined as the conscious awareness of where one learnt a particular piece of information, or indeed where one experienced a particular episode in one's life. An everyday example could be an inability to remember where something was put despite recollection of what is being looked for, or who told you something despite remembering what was said. In effect this viewpoint suggests that older people's item, or content, memory is relatively well preserved, the difficulty comes when the context that items were encountered in becomes important.

For instance, in the Hedden et al. (2001; 2003) experiments it may well be that older people suffer deficits simply because they can't remember which stage they learned the paired associates in. Indeed, Hedden and Park (2003) concluded that most of the variance in retroactive interference could be attributed to source monitoring errors rather than inhibitory failure since most of the interference associated with ageing could be explained by older participants' confusion between studied target word pairs and interfering word pairs read aloud. This interpretation is consistent with observed age-related deficits in context as opposed to item, or content, memories.

In an extensive meta-analysis Spencer and Raz (1995, see also Zacks, Hasher & Li, 2000), for instance, found a consistent age related deficit for contextual details relative to content memory. Furthermore, they found a greater age-related deficit for contextual details unrelated to stimuli, such as spatial location, time of day or position in a sequence, although this was less for stimulus attributes directly related to targets, such as their size and colour. In addition they found that age related deficits were markedly exacerbated for more effortful modes of retrieval (cued and free recall) than for recognition, and that more effortful encoding instructions led to a small but consistent increase in age differences for content memory.

Later research by Glisky, Rubin and Davidson (2001) showed that older people failed to initiate the processes that bind context with target stimuli during encoding. They found that older people could differentiate between a voice they had heard at study and a novel voice when oriented towards the voice by asking them to judge whether stimulus voices were likely to be heard on the radio. They could not, however, remember the sentences the voices had spoken in an identical forced choice task completed fifteen minutes later. A subsequent manipulation found, however, that older participants could not only remember the voice as in the first experiment, but could also recognise the sentences in the subsequent block when asked in this stage to rate how likely it was that the sentences would be spoken on radio.

A further consideration concerns whether older adults are disproportionately affected by the requirement to recall specific (e.g. particular person) or partial (e.g. person's gender) source information. Simons, Dodson, Bell and Schacter (2004) found age differences in both specific and partial source memory when participants were matched for recognition memory but no age differences in specific source memory when groups were matched for partial source memory. This implies a global deficit in the ability to bind context to memory irrespective of the level of detail required.

A note of caution, however, emerges from research conducted by Siedlecki, Salthouse and Berish (2005), who suggest that differences between content and context memory arise from the relative importance of remembering information rather than its source. Furthermore, they found a strong relationship between content and source memory in that those individuals who performed strongly on one measure were good at the other and that correlations between source memory tests were relatively weak. Lastly they report that relationships between source memory and measures of fluid intelligence, processing speed, and episodic memory were high. Taken together this implies that perhaps source memory may not be an independent construct that is particularly prone to the effects of ageing, although these data cannot deny that context memory is age sensitive and another interpretation could be that it is not surprising that source memory correlates with other age sensitive measures if this ability underlies much of the observed age related decline in cognitive abilities. Yet another interpretation could be that there may be a failure to inhibit inappropriate contextual details that leads to poor source memory, so Hasher and Zacks' (1988) theory cannot be ruled out entirely here.

Certainly it seems likely that the ageing process adversely affects memory for context, and this may have a bearing on associative learning.

Consider again a biconditional problem (AB+, CD+, AD-, BC-). Here responses to stimuli are contingent on the configuration of the elements. Another way to think about this is to suppose that each compound represents a separate context in which elements are found. If this is the case then older people may have special difficulties on problems with non-linear solutions, as opposed to linearly soluble ones such as the conditional problem, over and above the observed difficulties of younger participants. This may be because older participants may not initiate the necessary processes at encoding to enable them to differentiate the context in which elements had been presented in terms of both the presence of other stimuli and response associations. Alternatively, an inhibitory failure could still explain these data equally well since participants may be unable to inhibit inappropriate responses relating to similar stimuli.

Another demonstration of memory difficulties for older participants concerns prospective memory, or remembering to remember something. Since this is postulated to require considerable conscious effort it is no surprise that age related decline is found in this domain as well.

4.7: Prospective Memory

Prospective memory (PM) is a relatively recent area of investigation. Most paradigms ask participants to perform a distracter task during which they are required to remember to perform another relatively simple task (PM task), such as press a button, tick a box, or ask for something, either after a specified time (time based PM), or after a particular event (event based PM), or in a particular place (place based PM). Clearly this task requires WM since participants must remember to carry out the PM task at the same time as performing another task. Although it may be that remembering what the task was may require retrospective memory resources researchers have typically tried to minimise these requirements in PM tasks by making them as simple as possible (Maylor, 1998).

Other researchers (e.g. Kliegal, McDaniel & Einstein, 2000) have found no differences between older and younger participants in terms of retaining task instructions or task execution. Presumably PM also requires inhibition in that tasks must be maintained in WM whilst the distracter task is being performed yet not acted upon and vice-versa. Indeed West and Craik (2001) found that performance in PM tasks was mediated by measures of processing speed, working memory, and inhibition, although note that the DSST was used as the processing speed task and may therefore reflect higher cognitive processes (Parkin & Java, 1999, 2000).

Evidence suggests that time based PM tasks are more difficult than place or event based tasks, and that age differences are exaggerated in these types of PM tasks (Park, Hertzog, Kidder, Morrell & Mayhorn, 1997). This is consistent with Craik's (1986) hypothesis that those memory processes that require greater self initiated processing should suffer the greatest age-related deficits since both events or places could act as cues to retrieve the PM task, whereas simply having to remember to complete the PM task at a pre-specified time surely requires self initiated processing.

Further evidence is summarised by Maylor, Darby, Logie, Della Sala and Smith (2002). They review evidence gathered by Darby and Maylor (1998) that suggests that increasing task demands by switching the level of stimulus analysis between background and PM tasks exacerbated age differences. This study gave participants a target word and six possible responses for the background task, which was either structural or semantic. The structural background task required participants to write down the response word that followed the target word alphabetically, whereas the semantic task asked for the response word closest in meaning to the target word. Concurrent PM tasks were similarly semantic or structural. Here participants had to circle the trial number either if the target word was a colour word for the semantic task, or if the target word had a double letter for the structural PM task. Older people completed the semantic background task better than younger participants regardless of PM task, possibly because of their greater crystallised intelligence, although they were worse at the structural background task, presumably as this involved fluid abilities. Younger participants were overall better at PM tasks even when those who did not remember the PM task instructions were excluded. This superiority was particularly marked in conditions when there was a disparity between background and PM tasks, that is, when there was a semantic background task followed by a structural PM task or vice-versa.

Lastly, a more recent meta-analysis by Henry, MacLeod, Phillips, and Crawford (2004) illuminates a problem with laboratory based PM research. In this analysis laboratory based studies followed the pattern of results outlined above: older participants perform worse than their younger counterparts for both event and time based PM tasks, although a greater age related decline was found for free recall memory than PM. This is perhaps because free recall involves 'executive' processes in the form of purposeful behaviour, as demonstrated by Crawford, Bryan, Luszcz, Obonsawin, and Stewart (2000). It is also possible that older participants' poor performance was the result of a failure to inhibit similar stimuli or confusion over the sources of memories. Despite the mild surprise of the latter finding it is relatively standard compared to the observation that older participants perform better at naturalistic PM tasks than younger people. This finding may reflect the greater use of mnemonic strategies and prompts by older people only too aware of the fallibility of memory whereas the lack of aide memoirs such as calendars and notes in a laboratory situation demonstrates what happens when these aids are denied.

For PM overall, therefore, older people's difficulties are exacerbated by the lack of environmental support or cues in time based paradigms in particular and laboratory situations in general, suggesting that the deficit may be the result of problems with self initiated processing. Similarly the relative difficulty of task shifting deficits (Darby et al. 1998) may be the result of cognitive inflexibility, or a failure to change strategies when appropriate. Overall this seems to reflect Craik's (1986) hypothesis that performance in tasks low in environmental support and high in self initiated processing requirements will exhibit greater age related declines. Presumably this means that changes in any rules induced by participants in Shanks and Darby's (1998) studies should result in age related deficits, just as older participants should be more prone to retroactive and proactive interference in experiments similar to those conducted by Shanks et al. (Shanks, Charles, Darby, and Azmi, 1998; Shanks, Darby, and Charles, 1998).

One explanation of age related cognitive decline that links processing speed, WM, inhibition, attention, and prospective memory is the prefrontal cortex theory of cognitive ageing (West, 1996). This theory, as the name suggests, postulates that much observed age related cognitive decline can be explained by a deficit in processes associated with the prefrontal cortex.

4.8: The Frontal Cortex Theory of Cognitive Ageing

The view that the mnemonic deficits discussed so far may be associated with a physiological decline in the function of the frontal cortex is contingent on several strands of evidence. Firstly there is a need to establish whether the frontal cortex suffers a marked physical age related decline. Secondly one would presume that deficits in neuropsychological tasks associated with a loss of frontal function would also be suffered by an ageing sample of participants and the results of neuropsychological assessments to predict or be associated with performance on target tasks such as PM, WM, inhibition and source monitoring tasks.

In terms of the first point there is a plethora of evidence from histological and imaging studies to suggest that the prefrontal cortex suffers disproportionate physiological decline during normal ageing. Raz (2000) presents an exhaustive review of extant data, as does Woodruff-Pak (1997). Both authors point to global changes in the ageing brain in terms of decreased overall volume, cerebral blood flow, and an accumulation of lipofuscin (the 'wear and tear' pigment) and note that evidence for this is as equivocal as the notion of monotonic and universal age related cognitive decline in that individual differences seem to increase with age. Despite this both reviews conclude that frontal areas are selectively impaired with age, as are concentrations of dopamine within the prefrontal area. Indeed Raz (2000, p.37) described the prefrontal cortex as "highly vulnerable" to the adverse effects of ageing, and Woodruff-Pak (1997, p.92) concludes that "the prefrontal cortex is smaller in volume and less activated in older adults".

Other researchers have quantified whole brain atrophy in old age at 15%, whereas frontal areas exhibit an average 22% decline in volume (Shan, Liu, Sahgal, Wang & Yue, 2005). It seems, therefore, that the frontal lobe theory may be correct in assuming that there are structural differences between older and younger people's frontal cortices.

We now turn to the second question: do the patterns of deficits suffered by patients with frontal lobe damage and ageing participants converge? The Frontal Lobe is generally associated with the executive direction and control of cognitive processes, attention, memory, and planning. Recall that in Chapter 2 some of the deficits associated with Frontal Lobe (FL) damage were discussed. These included difficulties with planning sequences of actions, making decisions and using experience to guide future behaviour in everyday tasks (Shallice & Burgess, 1991; Bechara et al. 2000). Furthermore there are extant data demonstrating that FL patients were impaired in a range of laboratory tasks that imply a specific set of deficits.

Specifically difficulties have been found in the Towers of Hanoi problem, implying a deficit in the ability to formulate and execute plans involving multiple prospective and retrospective cognitive operations (Miyake et al. 2000). Further deficits are apparent in the WCST (Dempster et al. 1999), suggesting a vulnerability to perseverative errors or the failure to inhibit previously successful but no longer applicable strategies (Gunning-Dixon et al. 2003); the Stroop (1935) paradigm (e.g. Damasio, 1994), implying a problem with inhibition of task irrelevant stimuli, despite an ability to report a conscious knowledge of their redundancy (Duncan, 1995). Similarly FL patients tend to become confused between task relevant and irrelevant stimuli and have difficulty in consciously differentiating between stimuli with different outcomes (Henkel, Johnson, & De Leonardis, 1998; West, 1996; Dimitrov, Granetz, Peterson, Hollnagel, Alexander, & Grafman, 1999), and the appropriate allocation of processing resources in WM (Hartman, Pickering & Wilson, 1992). FL patients also have difficulties using a distinctiveness heuristic in that being asked or given the opportunity to encode extra details about stimuli does not facilitate episodic memory for this group relative to normal controls who benefit from extra information (Budson, Dodson, Vatner, Daffner, Black & Schachter, 2005). In terms of basic memory processes prefrontal areas are also involved in encoding and retrieval processes in episodic memory (Desgranges, Baron & Eustache, 1998; Gabrielli, 1998; Tulving, 2002) and FL patients have greater difficulty with free than cued recall and least problem with recognition memory (Gabrielli, 1998; Tulving, 2002).

In terms of similarities between age related and FL related cognitive deficits, measures based on perserverative errors, inhibition, content and context memory and source monitoring, WM, and PM have all been statistically associated with both age and frontal lobe function as assessed by neuropsychological instruments such as the WCST or in vivo scanning procedures.

Ridderinkhof, Span, and van der Molen (2002; see also Rhodes, 2004), for instance, demonstrated that older adults were prone to perserverative errors in WCST like tasks and that they were less able than younger adults to make use of cues that denoted a rule shift, suggesting inflexibility when dealing with new situations or ideas. Not only is this observation consistent with a frontal lobe explanation for cognitive ageing but also with declines in fluid, rather than crystallised intelligence.

Note, however, that perseveration is not synonymous with perseverance: Stuart-Hamilton and McDonald (1998) found that older adults persevered just as long as younger participants did in trying to solve the insoluble ‘Bridges of Konigsberg’ problem. Milham, Erickson, Banich, Kramer, Webb, Wszalek and Cohen (2002) found that decreased dorsolateral prefrontal cortex activity in older participants in an fMRI study of the Stroop task reflected their inability to implement attentional control through the inhibition of irrelevant stimuli relative to younger people. This was especially marked when conflicting colour information was present (i.e. the colour word name conflicted with the ink colour) relative to colour congruent (i.e. the colour word’s name and ink colour were the same) and was least noticeable for neutral conditions (neutral word). Clearly these data not only suggest that prefrontal areas are recruited for selective attention and the inhibition of prepotent responses but that greater task demands led to greater age related deficits, a familiar pattern of results associated with ageing.

Henkel, Johnson and de Leonardis (1998) investigated age differences in source monitoring. Participants were presented with either line drawings of objects or names of objects they were asked to imagine as a line drawing. They found that older adults’ scores on batteries of neuropsychological tests for frontal and medial temporal lobe function both correlated with source accuracy, or context memory, for whether objects had been seen or imagined, but not for recognition accuracy, or item memory, following a two day delay. After a fifteen minute delay results showed a correlation between source accuracy and the medial temporal lobe battery, interpreted as demonstrating that the longer 2 day delay led to increased task demands that recruited frontally mediated reflective processes. All participants tended to report having seen rather than imagined non-presented stimuli if they were perceptually or conceptually similar to seen items, thus demonstrating a confusion between similar items, and that older adults sustained a greater deficit even after performance on an old-new recognition task was controlled for, despite the fact that they were more likely to guess they had imagined an object. They suggest the data demonstrate dissociation between binding of feature and context, suggested to be medial temporal lobe dependent, and strategic, effortful retrieval involving evaluative judgements, suggested to be mediated by the frontal lobes.

Similarly Mather and Johnson (2003) present data to suggest that older people's story recall was more affected by schemas than younger participants'. Correlations between a frontal lobe battery and schema reliance and a medial temporal lobe battery and corrected recognition (items correct – false alarms) scores were statistically significant after partialling age out. Again this suggests dissociation between frontally mediated reflective evaluations of stimuli and medial temporally mediated binding of stimuli and story context.

Rhodes and Kelley (2005) found that scores on a battery of executive function test and age itself were better predictors of memory accuracy than scores on a speed of processing battery, suggesting that frontal lobe dysfunction is a better predictor of cognitive decline than speed of processing. Glisky et al. (2001) found that only older adults with below average frontal lobe function showed a source memory deficit, concordant with individual differences in cognitive decline with age (e.g. Rabbitt, 1993; Rabbitt et al. 2004) and consistent with their suggestion, alluded to earlier, that older adults fail to recruit contextual binding of stimuli due to impaired executive processes rather than, or in addition to, a deficit in contextual binding as such. The lack of medial temporal lobe battery mediation of source memory in the Glisky et al. (2001) study may be explained by task differences such that Mather and Johnson (2003) gave participants relatively simple tasks that presumably recruited fewer executive processes until sources had to be remembered over a long delay.

More evidence that only those older adults who suffer disproportionate FL deficits have greater memory deficits comes from a recent fMRI study that found older people with better declarative memory had right prefrontal activation greater than younger participants and equal left FL activation (Rosen, Prull, O'Hara, Race, Desmond, Glover, Yesavag & Gabrieli, 2002). Again this can explain differential rates of decline and the increase in variability of older people's ability to complete tasks involving executive abilities and episodic memory.

Given that WM is theoretically the component of memory that deals with conscious, strategic processing it is unsurprising that this is associated, or even synonymous, with frontal lobe function. As an example, Rypma, Prabhakaran, Desmond and Gabrieli (2001) gave a WM task consisting of encoding a one to six letter memory set, maintaining these letters for five seconds, and then deciding whether a probe letter had been part of the memory set. Again prefrontal cortex activation as assessed by fMRI was greater for younger than older participants, as was performance in the WM task, suggesting a frontally mediated decline in WM ability. Furthermore Salat, Kaye and Janowsky (2002) found age correlated negatively with WM, which was predictable with prefrontal cortex volumes, again suggesting an interaction between age and frontal function in determining WM abilities.

Since PM is theoretically a demanding form of memory it should not be unexpected that age related deficits on these tasks should also be associated with frontal lobe function. Despite this there are relatively few data available that explicitly compare age, PM, and frontal lobe functions. McDaniel, Glisky, Guynn and Routhieaux (1999) found that, of a group of older adults, those who scored highly on neuropsychological assessments of frontal lobe function performed better on an event based PM task, whereas no differences in PM performance were found between those adults who did well in a medial temporal lobe test battery.

Furthermore Martin, Kliegal and McDaniel (2003) varied the complexity of PM tasks and found that frontal function could not predict performance in a single task paradigm, but did in more complex tasks. Although age differences were apparent age itself only predicted performance in the most complex multiple PM task. These data suggest that frontal function is a good predictor of performance in PM tasks since PM itself involves the kind of self initiated processing associated with executive, frontally mediated processes. The clear difference between PM and source monitoring is that there seems to be little medial temporal lobe involvement in PM tasks, presumably since the retrospective memory components are designed to be as simple as possible. The inability of age to predict PM performance in all but the most complex tasks is consistent with the increase in variability of cognitive performance with age. While FL deficits, as well as general cognitive decline, may become more prevalent with age they are likely in many cases to be neither severe enough nor general enough to predict performance deficits in simple tasks.

Up until now it has been assumed that FL deficits are the result of structural changes. There is, however, some evidence to relate dopamine depletion in age and the loss of frontal function (see also Raz, 2000; Woodruff-Pak, 1997). Bäckman, Ginovart, Dixon, Wahlin, Halldin, and Farde (2000), for instance, conducted a positron emission tomography (PET) study to detect changes in the binding of dopamine receptors. Both age and dopamine binding predicted performance on word and face recognition tasks, and two perceptual speed tasks, with dopamine binding as the strongest predictor. Another PET study (Volkow, Gur, Wang, Fowler, Moburg, Ping, Hitzemann, Smith & Logan, 1998) found similar associations between age, dopamine levels, and performance on the WCST and Stroop tasks.

More recently researchers have found that dopamine binding in the striatum exhibits an age related decline that mediates decrements in executive function and episodic memory tasks (Erixon-Lindroth, Farde, Wahlin, Sovago, Halldin & Backman, 2005). Taken together these observations suggest that age related decline in executive abilities may not be solely due to physical deterioration or atrophy of brain tissue but that neurochemistry may also play a part. Another consideration is that the term 'frontal lobe function' is somewhat vague. Most commentators, including West (1996) tend to mean the prefrontal cortex when discussing changes in cognition with age. More recently researchers have made efforts to dissociate age effects in different regions of the prefrontal cortex. Lamar and Resnick (2004), for example found that neuropsychological assessments sensitive to orbitofrontal cortex damage showed greater age related declines than tasks assessing dorsolateral prefrontal cortex function, although remember that this area too seems to suffer structural decline (e.g. Raz, 2000) as well as being related to an age related deficit in inhibitory processes (Milham et al. 2002).

Recall, again from Chapter 2, that the orbitofrontal cortex (OFC) has been implicated in tasks requiring discriminations between rewards and punishments (Francis et al. 1999; Rolls, 1999, 2000, 2004; Thorpe et al. 1983) and discrimination reversals (Thorpe et al. 1983; Kringelbach et al. 2003). This, together with the evidence presented above, implies that OFC is not only vulnerable to age related decline but also that this decline may result in difficulties with predicting the consequences of one's actions and in amending responses following environmental changes, as in the WCST and other rule shift paradigms, and may therefore predict a vulnerability to proactive interference in reversal learning paradigms. Furthermore the observed age related decline in the dorsolateral prefrontal cortex may reflect an inability to inhibit inappropriate responses.

The predictions of neuropsychological theories of learning theories concerning the impact of FL dysfunction and dopamine depletion in the FL area have already been dealt with in Chapter 2. The weight of evidence discussed in this chapter suggests that the pattern of deficits associated with FL dysfunction is similar to the patterns of deficits associated with age-related cognitive decline. Furthermore scanning studies indicate that older participants are less likely to recruit frontal lobe areas when completing cognitive tasks. Given this the frontal lobe hypothesis is a strong contender for giving a physiologically grounded account for much of the observed age-related cognitive decline. One interpretation in the context of learning theories could be that older participants employ less effortful learning strategies based on elemental assumptions of summation, whereas younger people may have the capacity to choose strategies that best address a learning problem. For instance, it is clear from earlier studies (e.g. Shanks, Darby, and Charles, 1998) that younger participants can inhibit generalisation through perceptual similarity to a greater extent than even configural theories predict. This would involve effortful processes inhibiting responses to similar stimuli, as well as source memory for the exact configuration of stimuli that would indicate a particular learning context. In this view it is unlikely that older people would resist either proactive or retroactive interference to the same extent as younger participants and that their responses would be more consistent with elemental theories of learning.

Recall, however, from earlier in this chapter, that some of the age-related deficits in cognitive tasks were associated with compromised medial temporal lobe function. Specifically medial temporal lobe involvement is strongly suspected in explaining age-related deficits in associate learning (Dimitrov et al. 1999), source accuracy after a short delay (Henkel et al. 1998) and schema reliance (Mather et al. 2003). Furthermore ageing is associated with the decline in the volume and task related activity of hippocampal and medial temporal lobe regions (West, 1993; Woodruff-Pak, 1997; Raz, 2000), and many researchers doubt that the neuropsychological consequences of ageing are entirely attributable to deficits associated with a single brain area (see Band, Ridderinkhof and Segalowitz, 2002). Lastly, an increase in age is also associated with the depletion of acetylcholine (Woodruff-Pak, 1997; Raz, 2000; Terry & Buccafusco, 2003). This may result in difficulties in suppressing the autoassociative recall function of the hippocampal area and consequently result in deficits related to the storage of new memories and the establishment of stimulus-stimulus associations.

These observations and assumptions have more recently been formalised as the associative deficit hypothesis (Naveh-Benjamin, 2000; Naveh-Benjamin et al. 2002; Naveh-Benjamin et al. 2003; Naveh-Benjamin et al. 2004).

4.9: The Associative Deficit Hypothesis

Naveh-Benjamin (2000; Naveh-Benjamin et al. 2002; Naveh-Benjamin et al. 2003; Naveh-Benjamin et al. 2004) suggests that one explanation for the pattern of older adults' episodic memory deficits lies in their difficulties in creating associations between items and context as well as between items and other items.

Naveh-Benjamin (2000) tested the Associative Deficit Hypothesis (ADH) by comparing performance between item and interitem associations. In this series of studies older adults' memory for verbal material was tested by presenting word pairs and subsequently asking participants whether they had seen a particular word (item memory) and whether they had seen two items together (interitem memory). Older people performed worse overall but were disproportionately worse at recalling interitem associations. Similarly older adults performed more poorly when asked to identify which words were presented in which fonts despite a lack of difference between older and younger people in remembering either words or fonts separately. Furthermore the differences between old and young adults were exacerbated when recalling unrelated as opposed to related word pairs. This implies that the problem lies in encoding and retrieving novel word pairs rather than remembering existing relationships.

Naveh-Benjamin et al. (2003) replicated the earlier studies using nonverbal stimuli, in this case pictures. Again older adults were disproportionately worse on associative tests as opposed to item tests, whereas younger adults under divided attention simply showed a generalised performance deficit. This suggests that associative deficits with age are inexplicable in terms of attention and effortful processes, in turn implying that any associative deficit is unlikely to be frontally mediated.

Naveh-Benjamin et al. (2004) used face-name pairs to test whether older people's subjective complaints concerning name forgetting were due to simple forgetting or difficulties with the associations between names and faces. Each participant saw 40 face-name pairs and participants were then given forced choice recognition tasks for names, faces, and name-face pairs. Results showed no age differences in terms of face or name recognition but older participants were significantly less likely to correctly identify face-name pairs, even when a sub sample of older and younger participants were matched for item recognition. Furthermore young participants who completed the tasks under divided attention conditions were worst at item recognition, but were significantly better than older adults in terms of recognising face-name pairs.

Naveh-Benjamin (2000; Naveh-Benjamin et al. 2002; Naveh-Benjamin et al. 2003, Naveh-Benjamin et al. 2004) explain their results in terms of medial temporal lobe related deficits in the 'binding' of stimuli. This explanation certainly makes sense in terms of what we know of the function of this area and in terms of evidence to suggest atrophy and reduced function of this area in the later years of life. A note of caution is warranted at this point though, since there are no data in these studies derived from neuropsychological testing or in vivo scanning techniques to test the assumption that medial temporal dysfunction best explains these data. Although this assumption is entirely plausible it may equally be difficulties related to the conscious discrimination between existing stimulus representations that may cause the deficits observed by Naveh-Benjamin (2000; Naveh-Benjamin et al. 2002; Naveh-Benjamin et al. 2003; Naveh-Benjamin et al. 2004). Although FL dysfunction may not easily explain why inter-item associations are disproportionately affected, WM and attentional or inhibitory processes could explain why younger adults under divided attention conditions showed a different pattern of results. It seems that the associative deficit hypothesis may offer a reasonably coherent, albeit weaker alternative to the frontal lobe theory and, as with the frontal lobe theory we can draw on the predictions made by the neuropsychological theories of learning discussed in Chapter Two to anticipate the effect of ageing on associative learning. Caution over the interpretation of these data is, however, appropriate since the possibility of a failure of inhibitory or source monitoring processes has not been ruled out, and it may be that learning interitem associations represents a form of context learning similar to source monitoring deficits that appear to be mediated by declines in executive abilities associated with the frontal lobes.

One problem this leaves us with is in distinguishing between the effects of deterioration in processes associated with the frontal or medial temporal lobes. This is, however, assuming that the frontal lobe theory and the associative deficit hypothesis are in competition and that either theory can on its own explain all age-related declines in learning and memory. This competitive view may be disingenuous since neuropsychological and in-vivo scanning evidence suggests that both areas may be subject to structural and neurochemical decline. It may be more useful, therefore, to see these views as complementary and work towards delineating the extent of decline associated with each area and specifying the relative contribution each makes to age-related cognitive change. On balance, however, West's (1996) frontal lobe hypothesis seems more compelling since there are many more data to support this view that have been collected by many different research groups. Naveh-Benjamin's (2000) theory, on the other hand, lacks direct empirical support from outside of his own research group and is, therefore, a more speculative, although potentially useful, addition to the explanations for age related cognitive decline.

4.10 Directions for Research

From the literature that has been reviewed so far it has been possible to gain an insight into both associative learning and cognitive ageing. A multitude of factors seem to influence cognitive ability in later life, although this shouldn't be a surprise since by definition older people have had more time to absorb the experiences and environmental influences that, at least in part, shape our behaviour. Certainly it will be necessary to gather data that account for the health, lifestyle and general cognitive factors discussed over the last two chapters that may affect contingency learning abilities. Beyond this, however, it should be obvious to the reader that the clearest predictions concerning developmental changes in contingency learning abilities may be derived from a combination of observations of neuropsychological and cognitive deficits associated with age and theories of learning.

In terms of testing the nature of age related declines in learning the current research will adopt the food allergy paradigm employed by Shanks et al. (Shanks, Charles, Darby & Azmi, 1998; Shanks & Darby, 1998; Shanks, Darby,

& Charles 1998). Furthermore the experiments will employ positive patterning problems (PPP), negative patterning problems (NPP), as well as biconditional and conditional designs in order to illuminate any age related deficiencies. From what has been discussed so far it should be clear that both frontal (FL) and medial temporal (MTL) lobe deficits should result in learning deficits for the older participants. In some instances it may be possible to discriminate between the predictions offered by the FL theory and the associative deficit hypothesis.

FL theory would predict that NPPs should be more difficult than PPPs since the latter class of problem is soluble through summation, which surely requires fewer processing resources than acquiring a truly non linear discrimination (c.f. Experiments 1-5) and does not assume any inhibition of responses to similar stimuli. One can anticipate, therefore, that older people's learning will conform more closely to the predictions of elemental theories when asked to solve NPPs and biconditional problems. This should, in this instance, be due to difficulties remembering which of two similar stimuli lead to a certain outcome and in inhibiting inappropriate responses.

In addition it might be expected that FL theory would define 'complexity' in terms of increased strategic processing demands and WM loads. In this view using more stimuli, or exemplars, increasing the number of possible responses, or presenting different problems concurrently would exacerbate age related deficits (c.f. Experiments 1-8). The deficits predicted by FL theory should be the result of the use of simple elemental strategies in response to increasing processing loads. The resulting overgeneralisation should in this instance be analogous to Lashley and Wade's (1946) view discussed in Chapter Two: that overgeneralisation is the result of confusion between whether a stimulus leads to one outcome or another and that the failure to inhibit inappropriate responses may lead to erroneous predictions, rather than a consequence of confusion between stimuli. All of which should mean that problems with multiple responses should be more 'complex' than those with single responses, given that the number of exemplars stays the same (c.f. Experiments 1-2).

Equally, concurrent PPPs and NPPs presented together should be more 'complex' than two concurrent PPPs or two concurrent NPPs, and therefore lead to greater age related deficits (c.f. Experiments 3-5). Beyond this a relative inability to learn initial discriminations should lead to fewer rules of conjunction being induced among an ageing population (c.f. Experiments 2-5).

Furthermore, an ageing population should be more prone to perserverative errors if rules are changed or shifted, whereas a younger population should adapt to this change more flexibly (c.f. Experiments 9-10). Lastly an ageing population should be more prone to retroactive interference since a deficit in strategic processing resources will make it less likely the context in which initial learning took place will be identified correctly, and that even if it is initial learning will be more likely to be confused with later learning in any event through an age related deficit in generalisation processes through the inability to inhibit inappropriate responses (c.f. Experiment 11).

A purely MTL based deficit would, on the other hand, make slightly different predictions although would similarly anticipate overgeneralisation. Here this would result from an inability to distinguish between similar stimuli due to the kind of spreading activation advocated by Pavlov (1927) and Hebb (1949), again referred to in Chapter Two. Since both PPPs and NPPs involve the same level of 'complexity' in terms of creating and distinguishing between elements and their compounds a purely MTL deficit would predict little difference between the two, although compound learning should be more difficult than elemental learning (c.f. Experiments 1-5) and post experimental compound recognition should be impaired (c.f. Experiments 9-11). On the other hand conditional problems may still be seen as less ‘complex’ than biconditional problems, since their solution is contingent on learning that a single element signifies a response and another single element signifies no response (c.f. Experiments 6-8).

In terms of rule learning an MTL deficit implies that NPPs and PPPs couldn’t be learned and therefore no rule of conjunction could be induced. In this instance a rule shift would make little difference since there would be no rule to ‘unlearn’. Here ‘complexity’ can be viewed as the number of compound stimuli to be formed, rather than as the number of responses or in terms of processing demands (c.f. Experiments 1-5).

What the reader may note at this juncture is that both explanations alluded to above implicate overgeneralisation in their predictions for how learning ability may change with age. What is to be avoided is mere confirmation of Perfect and Maylor’s (2000) ‘dull hypothesis’ in detecting only a simple, monotonic decline in learning ability with age. Experiments will therefore be designed to address the questions outlined above in order to examine the role of changes in generalisation and rule induction processes with age. Before going on, however, some studies reported in the last few years will be considered to see if they can shed any light on the question of changes in associative learning processes with age.

Few extant data directly address the question of human ageing and stimulus-response learning to guide the present investigation. Bellebaum and Daum (2004) investigated age differences in eyeblink conditional discrimination using a paradigm identified as being sensitive to Medial Temporal Lobe damage (Daum, Channon, Pokey & Gray, 1991). In this study participants were exposed to two different coloured lights (A or B) that predicted whether a tone (S) that followed predicted an airpuff US (AS+) or not (BS-). They found that older participants were less likely to develop a CR to the AS+ compound relative to the BS- stimuli, demonstrating that older participants are slower to learn stimulus response discriminations. The problem itself is linear, so this could merely reflect a general, monotonic decline of cognitive ability with age, although it may be that younger participants were better able to recruit configural processes to facilitate quicker solution. This would make sense since the tone that immediately precedes the US was the same in each condition, and it would take longer for an elemental model to derive correct predictions because the model would have to learn that the tone stimulus was non-predictive, allowing the coloured lights to gain all the associative strength. A configural model, on the other hand, would assume that AS+ and BS- were separate stimuli, and although some generalisation should occur this would not be to the extent assumed by elemental models. Again, it seems useful to assume that humans make elemental predictions when configural processes break down and that this would lead to greater generalisation between similar stimuli.

Bellebaum et al. (2004) also found that younger participants' CR rate decreased over an extinction block, whereas older participants' CR rate did not. This may seem odd at first, and difficult to explain since this appears to imply some kind of deficit in younger participants due to accelerated decay of memory traces. A reconsideration of this observation in terms of West's (1996) FL theory may make more sense, though. By definition extinction trials are a change in the experimental conditions from reinforced trials so the decrease in CRs can be seen as flexible, adaptive responses to changing circumstances rather than symptomatic of decay. Equally, older participants' slower adaptation to environmental change could be characterised as a perserverative error and therefore consistent with FL decline (e.g. Dempster et al. 1999; Gunning-Dixon et al. 2003). Although Bellebaum et al. (2004) reported no age group differences in reported awareness of contingencies there was an interaction in that aware older participants exhibited fewer CRs than their younger counterparts whereas there were no age differences among participants who were unaware of the contingencies, suggesting that older participants were less able to make use of explicit knowledge than younger people, suggesting a deficit in conscious, reflective processes. This could relate to rule learning in associative learning (Shanks & Darby, 1998) in that some younger people may have been quicker to learn initial contingencies (possibly as a greater ability to recruit configural processes) and, consequently, induced applicable rules more easily.

Lastly Bellebaum and Daum (2004) report that discrimination and CR frequency correlated with measures of memory intended to reflect an MTL deficit but not with any measures from a battery of FL tests. This indicates that age related declines in associative learning are perhaps due to problems discriminating between CRs as a result of impaired stimulus-stimulus learning, although it should be noted that this experiment's design was a fairly simple discrimination that may not have tapped into higher order executive processes sufficiently for a decline of this nature to be detected.

Mutter and Williams (2004) asked older and younger participants to judge whether pressing a spacebar in a computer task caused a triangle to flash on screen where the probability of this occurring was varied between –0.8, -0.4, 0, 0.4, and 0.8. Participants were asked to judge the probability on a percentage scale from –100 to +100 in three conditions: short and long interval 60 trial conditions and a short interval 240 trial condition. They were also offered a financial reward for correctly identifying the probability of a spacebar press stopping the triangle from flashing. Although participants completed background tests of verbal fluid and crystallised intelligence, executive ability, and associative memory these were, unfortunately, not analysed with respect to experimental results but showed the expected age related differences. Older adults were found to be especially poor at estimating negative contingencies when there was a short interval between response and outcome, suggesting a potential difficulty with creating stimulus representations in short time periods, although this trend was apparent in young adults too. Again this is, relative to the following experiments, a simple task and it is difficult to extrapolate from these results, especially in the absence of any background data comparisons.

Mell, Heekeren, Marschner, Wartenburger, Villringer and Reischies (2005) investigated the effect of ageing on stimulus-reward association learning using a probabilistic object reversal task (pORT) and test batteries reflecting general cognitive and executive abilities. The pORT involved presenting participants with four of six stimulus letters on each trial and choosing the letter associated with the greatest non-monetary reward (either 40, 20, 0, -20, or –40 points) to maximise the number of points gained over the task as a whole. Once participants had reached a learning criterion and had identified the maximum reward available over 6-8 successive trials the feedback schedule was changed without warning.

Results showed that younger participants were more successful at the task overall, needed fewer trials to reach criterion accuracy, and made fewer random errors. A surprising finding, however, was that older adults made fewer perseverative errors, that is they persevered no more than younger people with incorrect responses and pORT performance was correlated with only one of a battery of executive tasks (the Self Ordered Pointing Task, Petrides & Milner, 1982). Analysis of relationships between pORT and general intellectual ability and processing speed were non-conclusive. The lack of relationships between associative learning and general and executive abilities may, again, be a reflection of the simple nature of the task. In the pORT participants merely have to identify one of six letters associated with maximal reward rather than learn multiple stimulus response contingencies and the task is therefore linearly soluble.

Other strands of research have more directly addressed the question of whether generalisation processes are affected by age. Wearden, Wearden, and Rabbitt (1997) investigated time perception with older adults split into Young Old (60-69) and Old Old (70-79) age groups. Experiment 1 was a direct replication of one conducted by Wearden (1992) on undergraduates, so these results were used as an informal comparison. Participants were presented with a 400ms standard stimulus and asked whether subsequent stimuli of 100, 200, 300, 400, 500, 600, and 700ms were of the same duration by responding with ‘Yes’ and 'No' keys on a computer. Each trial was followed by corrective feedback and so could be considered to be analogous to a learning task. Wearden (1992) had found that young adults' responses were analogous to rats' in that the distribution of 'Yes' responses in that by far the majority were to 400ms duration stimuli, with responses falling sharply as stimuli became less similar than the standard, although responses to longer durations were more frequent than to shorter durations, leading to an asymmetrical distribution of the frequency of responses. Wearden et al. (1997) found an increasing and significant tendency across the two older age groups toward a flatter, more symmetrical distribution of 'Yes' responses, indicating that generalisation between stimuli increased with age. They also found that IQ and age predicted the frequency of incorrect responses such that increasing age and decreasing IQ were synonymous with increasing inaccuracy and, as such, also predicted the extent of overgeneralisation.

Experiment 2 used a temporal bisection task indicated no age differences in Young-Old and Old-Old participants' ability to state whether 300, 400, 500, 600, and 700ms stimuli were closer in duration than 200ms and 800ms stimuli. Furthermore, data were almost identical to those obtained by Wearden (1991) from undergraduates, overall indicating that generalisation processes may be distinct from discrimination processes in terms of age related change.

These results were replicated by McCormack, Brown, Maylor, Darby, and Green (1999) with the exception that older adults' generalisation gradients were similarly asymmetrical to, though flatter than, the younger groups', and IQ no longer predicted response accuracy significantly, although Digit Symbol Substitution Task (Wechsler, 1981) scores did. McCormack et al. (1999) also found no differences between age groups in terms of a temporal bisection task, reinforcing the notion that generalisation and discrimination processes are differentially affected by age.

This leads to the question of whether generalisation processes are, as suggested by Gluck and Myers (1993, 1997), mediated by unconscious stimulus-stimulus processing in the MTL region or whether generalisation processes are contingent on more conscious, reflective processes consistent with FL region processing. Recall that Bellebaum et al. (2004), in an eyeblink conditioning task, found that discrimination learning correlated with MTL test battery scores but that age differences only manifested themselves among those who reported conscious awareness of contingencies. This suggests that a decline in conscious, reflective processes were also at work and may have been the major source of age differences in that experiment. This observation may relate to the dissociation between discrimination learning and generalisation processes in the experiments of Wearden et al. (1997) and McCormack et al. (1999), and suggest that the latter may be directed more by conscious, executive processing than by unconscious stimulus-stimulus learning.

More evidence that generalisation processes may suffer age related decline and are associated with frontal lobe deficits comes from LaVoie, Willoughby, and Faulkner (2006). They employed a false memory paradigm to test Frontally Impaired (FI) and Older Control (OC) groups of older adults and compare their performance to younger participants'. They used twelve established semantic category lists of fourteen words (e.g. fruits, clothing) and took away the two words most highly associated with the category (e.g. apple, pear) and the two words least associated with the category (e.g. melon and nectarine) to use as strongly and weakly associated critical non-presented lures (Strong CNP and Weak CNP). The twelve lists of the remaining ten words in each category were presented to participants before they were asked to complete a recognition task in which list items, CNPs, and filler stimuli were presented and participants had to state whether they had seen the stimuli before or not.

False recognition of Weak CNP items showed no significant differences between Young, OC, and FI groups, suggesting that if items are sufficiently semantically different both age and frontal lobe function made little difference. False recognition of the Strong CNP stimuli did, however, show age differences such that older participants in general were more likely to falsely identify an item strongly associated with the semantic category of a studied list as having been seen than younger participants, and that the FI group were particularly prone to this tendency. This suggests, again, that generalisation gradients become flatter with age and that frontal lobe impairment may underlie this deficit.

In a related experiment Badre and Wagner (2005) also found that fMRI analysis demonstrated frontal region involvement in a task involving judging whether a probe word had been in a recently presented set of target words. They found that participants made more errors, and took longer to make a judgement about words that had been presented in a recent, but not the most recent, target list, and that such judgements engaged more frontal area activity. Again, this suggests that the closer an item is in time or meaning to another item, the more likely people are to become confused between items and that this tendency towards interference increases with age and is negatively associated with executive abilities and extent of frontal activation.

Given the convergence between generalisation, frontal lobe ability, and age it seems reasonable to suppose that generalisation as a process may be crucial in establishing the nature and extent of any age related deficits in associative learning processes. Furthermore, given that the Gluck et al. (1993, 1997) model emphasises generalisation as a hippocampal or medial temporal lobe ability it is questionable how useful the predictions of this theory will be in establishing a reason for any observed age related changes.

Fortunately it is apparent that overgeneralisation in learning would be more consistent with elemental than configural processing. This means that a simple way to judge whether participants are overgeneralising would be to compare behavioural data with the predictions of elemental and configural models of learning.

Since data concerning ageing and associative learning have been derived from experiments of a relatively simple design the current research will seek to firstly describe the nature and extent of any age related deficits in contingency learning in NPP, PPP, conditional and biconditional problems before going on to examine the consequences of rule shifts and attempts to induce retroactive interference. The results of these studies will make it possible to offer tentative conclusions on the likely neuropsychological bases of contingency learning deficits, test the predictions concerning generalisation processes, and to indicate directions for further, more specific research.

The remainder of this thesis will be structured as follows. The next chapter will look at the individual differences factors identified in previous chapters. The purpose of this analysis is twofold. Firstly one may ascertain whether the current samples of older and younger adults exhibit a similar pattern of differences to that seen in other samples. Secondly it will be possible to identify those factors that vary with age and may have a bearing on general cognitive ability and therefore may predict overall performance across cognitive domains. This will allow either control or analysis of the effects of background factors on conditional learning.

Following this a series of experiments will be described and analysed allowing conclusions concerning the nature and causes of any age related decline in contingency learning ability to be drawn and directions for future research outlined. Experiments 1 to 5 deal with positive and negative patterning problems whilst Experiments 6 to 8 will examine conditional and biconditional problems. These experiments will manipulate the number and nature of the problems systematically in order to ascertain the extent of age related learning and rule induction deficits, and whether older participants' learning is more consistent with elemental or configural processes of generalisation in relatively simple one stage learning tasks. Experiments 9 to 11 will go on to use multiple stage experiments to examine the effects of pro- and retro-active interference on learning, and again whether this implies if participants are using elemental, configural, or rule based processes of generalisation.

Chapter 5: Individual Differences Measures and Results

5.1: Individual Differences Measures

In order to estimate how far cognitive function in a specific domain is compromised by age it is important to consider factors that co-vary with age but are also associated with general cognitive decline. Factors that may underlie age related cognitive decline other than age itself have already been discussed in Chapter 3, but include education, 'processing speed', general intelligence, basic memory, motor speed and hand-eye co-ordination, health, and lifestyle. This section revisits some of the evidence for these factors, as well as detailing the measures that have been used to account for them in the present research.

Participants themselves were split into three age groups. The Young (Y) group were 279 undergraduates who completed selected background assessments as part of a course requirement, and later participated in experiments either voluntarily or as part of their courses. Older participants were all independent, community dwelling volunteers, and were split into two age groups: the Young-Old (n = 164: YO: 55-74), and the Old-Old (n = 59: OO: 75 and over). All of the older participants were recruited over a period of a year through posters, newspaper articles, radio interviews, and contact with older people's groups. All were spoken to personally by telephone on volunteering and the nature of the investigation explained to them before they attended any assessments.

Those that decided they might like to participate were then sent a questionnaire and informed consent form by post to complete if and when they chose. Most participants who received a questionnaire completed and returned it, although some decided not to continue. Data from completed questionnaires were entered into a spreadsheet and hard copies kept in a locked filing cabinet, as were experimental data. Personal details were entered into a database used only for contacting participants and cross referenced with questionnaire data by assigning each volunteer a participant number.

Following questionnaire return participants were invited to either a University of Wolverhampton campus in Wolverhampton or Telford, or alternatively venues were arranged at local centres in Oswestry, Leek, Bradley, Ludlow, and Dudley. The reasoning behind this was that participants from outlying areas would be more comfortable travelling to a local centre than to the University itself. It also allowed participants to be recruited from a wider geographical area. Participants were offered no inducements beyond refreshments following background and experimental sessions, as well as the opportunity to ask questions.

The questionnaire sent to participants was an important source of background data. Participants answered questions pertaining to many of the factors discussed in Chapter 3, which are discussed more fully below. One of the most reliable predictors of cognitive ability in old age is participant's level of education. A simple 'years of education' factor can account for much of age related cognitive decline in many cases (e.g. Salthouse, 1991), and participants were asked what level of education they had attained and how old they were when they left full time education.

Another important issue is that of the relationship between declining health and increasing age. As the brain is part of the physical body it makes sense that somatic illness should have an adverse effect on cognition. The questionnaire asked for general information such as recent hospitalisation, and number and nature of prescribed drugs, as these factors may have indicated the presence of serious illness. Participants also rated their general health on a five-point scale, and were asked to indicate the extent to which any health problems had a negative impact on social and necessary activities.

Some more specific disorders commonly found to increase with age and which adversely affect cognition are covered as well. Zelinski, Crimmins, Reynolds, and Seeman (1998) found that diabetes, high blood pressure or stroke had a negative impact on basic cognitive abilities, while Barusch, Rogers, & Abu-Bader (1999) found similar results for depression. Van Boxtel, Buntinx, Houx, Metsemakers, Knottnerus, and Jolles (1998) found cognitive decline in older people exacerbated by heart disease, circulatory disorder, and bronchitis or other respiratory disorder while Streisand, Rodrigue, and Sears (1999) found evidence relating liver and kidney disease to cognitive decline. Finally Ebert and Heckerling (1998) related cancer and Parkinson's disease with cognitive deficits, as well as highlighting glaucoma or cataracts' negative impact on participants' ability to communicate. Participants were asked on the questionnaire whether they had suffered from any of these disorders.

Finally the extents of daily activities need to be considered. Pushkar, Arbuckle, Conway, Chaikelson, and Maag (1997) found that participant scores on the Everyday Activities Questionnaire (EAQ) accounted for some age related variance in cognitive ability. The EAQ itself is divided into Necessary (e.g. using a car or public transport to get out) and Voluntary (e.g. reading, gardening) components as well as generating a total score by asking participants the frequency with which they participated in the activities asked about. In addition to an amended version of the EAQ participants were asked about the frequency of their social interactions with family and friends, and whether they live alone or with a partner. Hopefully these data will provide an estimate of the influence of keeping active and sociable into older age on cognitive abilities.

In addition to questionnaire data several more formal tests of cognitive, visuo-spatial, and sensori-motor ability were undertaken. Salthouse (1996) suggests that a general slowing of the brain's function can account for age related cognitive decline. This 'processing speed' factor was accounted for with a simple test of perceptual speed, the digit cancellation task (Parkin & Java, 1999, 2000). This task involved giving participants a 20x20 grid of single digits between 0 and 9. Within this grid were 40 number 4s, and participants were asked to mark as many number 4s as they could within thirty seconds.

General intelligence can be split between fluid and crystallised intelligence. Fluid intelligence is suggested to reflect the ability to operate on new information, solve problems, and think flexibly. Most large-scale studies show that fluid intelligence declines with age (e.g. Horn & Masunaga, 2000). In common with many studies of cognitive ageing the Alice Heim 4 group test of general intelligence (AH4, Heim, 1968) was used as a measure of fluid intelligence. In this instance the standardised instructions for this timed test were closely adhered to, as detailed in the handbook. The AH4 itself is divided into Verbal and Spatial components that are summed to derive an AH4 Total score. The verbal component is composed of assessments of verbal and numerical problem solving such as verbal analogies and number series. The spatial component requires participants to solve problems involving spatial relationships and the mental manipulation of objects.

Crystallised intelligence represents participants' level of acquired knowledge and was assessed with the equally ubiquitous Mill Hill Vocabulary Scale Form A (MHV, Raven, 1982). The MHV is an untimed assessment that presents participants with a series of increasingly obscure words and asks them to identify synonyms from a list of six alternatives.

The Digit Cancellation task, AH4, and MHV were completed by both undergraduates and older volunteers. In addition older volunteers completed two further tasks. Evidence for age related decline in short-term memory is equivocal (e.g. Craik & Jennings, 1992) but any decrement would adversely affect participants' performance on virtually any cognitive task.

An estimate of short-term memory capacity was made using Wechsler's (1955) forward digits task. The task was administered to participants as a group; participants were read out strings of digits varying in length between three and nine digits long. They were then asked to remember the strings immediately after they had been presented and record their responses in an answer booklet. Their digit span was the longest string of numbers they could remember in the order presented without mistakes.

Motor speed and hand-eye co-ordination are of obvious importance in completing any cognitive task in which time is a factor. It has been suggested that some of the age-related differences in fluid intelligence may be attenuated by removing time limits, and any experiment involving response times should take account of physical slowing and co-ordination problems. These factors were assessed using the MacQuarrie Test for Mechanical Ability (McQ, MacQuarrie, 1953), a standardised battery of motor and visuo-spatial function. Four timed assessments were used from the battery: Tracing, Dotting, Tapping, and Copying and the scores combined to give a total. The ‘Tracing’ test presented participants with a series of vertical lines with gaps in varying positions. Participants had to ‘trace’ a horizontal line through the gaps without touching the vertical lines. ‘Dotting’ involved placing a dot in the middle of a series of small circles without touching the edges. ‘Tapping’ required participants to place three dots in the middle of a series of larger circles, although speed was more important in the latter than accuracy. In both instances scores were given by the number of valid dots. Finally ‘Copying’ required participants to copy a complex figure into a dotted grid. Scores here were the number of correct lines copied. All McQ tests were subject to time constraints and administered according to the standardised procedures given in the handbook.

5.2: Individual Differences Results

In common with statistical analyses throughout this thesis the probability value accepted as showing a statistically significant result is $\alpha < 0.05$. This section examines the descriptive data gathered from questionnaires and background tests to assess whether the population of participants can be regarded as representative of the populations from which they are drawn. Furthermore it is hoped that factors will emerge that may help to explain age differences in general cognitive ability. These factors may then be used as predictor variables in later multiple regression analyses.

Table 5.1 (below) shows Means and standard deviations (S.D.) of each of three age groups for measures of age, years of education, AH4, MHV, and digit cancellation scores. The values show that measures of fluid intelligence (i.e. AH4 scores) decreased with age, as did the Digit Cancellation scores. Crystallised intelligence (i.e. the Mill Hill Vocabulary scale: MHV) increased with age. These results are consistent with the literature, suggesting that our samples are representative.

Table 5.1: Means and Standard Deviations For Age and Individual Differences Variables by Age Group

Age Group	Young			Young-Old			Old-Old		
	Valid N	Mean	S.D.	Valid N	Mean	S.D.	Valid N	Mean	S.D.
Age	279	23.69	7.01	164	66.78	5.30	59	79.46	3.75
AH4 Verbal	279	35.72	8.31	75	33.63	9.12	30	31.17	8.99
AH4 Spatial	279	48.31	10.14	75	36.37	10.36	30	29.80	8.66
AH4 Total	279	84.03	16.95	75	68.73	19.05	30	60.97	16.67
MHV	279	26.68	3.66	79	32.78	4.63	31	34.03	4.28
Digit Cancellation	279	23.48	5.41	75	16.89	4.10	30	15.97	4.32
Years of Education	279	14	0	163	12.4	2.88	59	12.07	3.02

One way ANOVAs and Bonferonni post hoc tests were performed to ascertain whether these group differences were significant. ANOVA showed significant differences in terms of AH4 Verbal ($F_{(2,381)}$=4.995, p<0.008), AH4 Spatial ($F_{(2,381)}$=76.21, p<0.001), AH4 Total ($F_{(2,381)}$=40.98, p<0.001), MHV ($F_{(2,386)}$=107.97, p<0.001), Digit Cancellation ($F_{(2,381)}$=69.03, p<0.001), and Years of Education ($F_{(2,498)}$=47.301, p<0.001). Bonferonni tests showed that these differences were significant between Y and OO groups for AH4 Verbal (Mean Difference = 4.55, p<0.02), AH4 Spatial (Mean Difference = 18.51, p<0.001), AH4 Total (Mean Difference = 23.06, p<0.001), MHV (Mean Difference = 7.35, p<0.001), Digit Cancellation (Mean Difference = 7.51, p<0.01), and Years of Education (Mean Difference = 1.93, p<0.001).

Differences were also observed between Y and YO groups in terms of AH4 Spatial (Mean Difference = 11.93, p<0.001), AH4 Total (Mean Difference

= 15.3, p<0.001), MHV (Mean Difference = 6.1, p<0.001), Digit Cancellation (Mean Difference = 6.58, p<0.001), and Years of Education (Mean Difference = 1.595, p<0.001). Additionally there were significant differences between YO and OO groups in AH4 Spatial (Mean Difference = 6.57, p<0.009).

Comparisons were also made between YO and OO groups in terms of data gathered from the questionnaire sent to all participants on recruitment (see section 5.1). Table 5.2 (below) shows Medians and Inter Quartile Ranges (IQR) for ordinal level data derived from the questionnaire. Subsequent analysis was by Mann-Whitney U test.

Table 5.2: Medians and Inter Quartile Ranges of Non-Parametric Questionnaire Data by Young-Old and Old-Old Age Groups

	Young-Old			Old-Old		
	Valid N	Median	IQR	Valid N	Median	IQR
Health Limits	N=163	5	1	N=59	4.50	1.5
Social Contact	N=162	3	0.5	N=58	3.25	0.5
EAQ Necessary	N=163	14	2	N=59	13	3
EAQ Voluntary	N=163	56	10	N=59	53	12
EAQ Total	N=163	71	11	N=59	70	13
Self Rated fitness	N=163	4	1	N=59	3	1
Self Rated Memory Decline	N=163	2	1	N=59	2	1
Units of alcohol	N=162	2	2	N=59	2	2
Self Rated Health	N=163	4	1	N=59	4	1
Hearing Quality	N=160	3	1	N=59	3	1
Vision	N=163	4	1	N=59	3	1

Health limits was a composite measure of two questions: How much difficulty do you generally have doing your usual everyday activities and tasks, both inside and outside the house because of your physical and emotional health; and does your physical and emotional health limit your social activities with family, friends, neighbours or groups. Both questions were answered on a five point Likert scale from ‘extremely limited’ (1) to ‘not at all limited’ (5). The overall ‘health limits’ measure was an average of these two scores, with low scores being more desirable. Social contact was an average of four responses: How often do you see your family; and how often do you see your friends (both measured on a five point Likert scale from (1) ‘less than once a year’ to (5) ‘daily’); please indicate how often you attend club/society activities (six point scale from (0) ‘never’ to (5) ‘daily’); and do you live: (1) alone; (2) with professional carers; (3) with friends; (4) with family; and (5) with a long term partner. Puskar et al.’s (1997) EAQ has already been described. ‘Self rated fitness’ was measured on a five point scale from (1) ‘very poor’ to (5) ‘very good’, and memory decline was measured on a six point scale from (0) ‘none’ to (5) ‘a great deal’. Units of alcohol was given as a five point scale from (1) none, through (2) less than 7, (3) 7-14, (4) 14-28, to (5) over 28. Self-rated health, hearing quality, and vision were given on a five point scale from (1) very poor to (5) very good.

For ‘Health Limits’ the OO were significantly more limited than the YO ($U (N_1=163, N_2=59) =3342.5, p<0.001$), although both groups scored highly on this measure. The OO group’s median EAQ Voluntary was significantly less than the YO ($U (N_1=163, N_2=59) =3802.5, p<0.02$), as was EAQ Necessary ($U (N_1=163, N_2=59)=3842.5, p<0.02$), and Total ($U (N_1=163, N_2=59)=3754, p<0.015$); but their Social Contact ratings were not significantly different. This tells us that the over 75s in our sample participated in fewer activities, made fewer necessary journeys, but saw as much of friends and family as the YO group. The YO group rated themselves as fitter ($U (N_1=163, N_2=59) =3960.5, p<0.04$), healthier ($U (N_1=163, N_2=59) =3342.5, p<0.001$: mean YO=3.9, OO=3.61), with better hearing ($U (N_1=163, N_2=59) =3342.5, p<0.001$), and sight ($U (N_1=163, N_2=59) =3568.5, p<0.003$). This pattern of results is unsurprising and it is certainly possible that the OO group's subjectively poorer health may be responsible for at least some of the variance in the observed age differences in terms of cognitive tasks. Alternatively it may be equally possible that differences in questionnaire scores may be due to respondent bias. Certainly those who took prescription medicines were significantly older ($t(219)=4.13$, $p<0.001$), and had worse visuo-spatial ability according to the MacQuarrie (1953) Tests ($t(107)=2.27$, $p<0.03$) than those who did not, perhaps indicating greater physical decline for this group.

Parametric measures restricted to the older sample included Digits Forward, Years of Education, Number of Prescription Medicines (NPM), and the MacQuarrie Test for Mechanical Ability (MCQ). Table 5.3 shows the means and standard deviations for these measures for YO and OO age groups. Only the YO group's advantage for MCQ Total proved significant ($t(108) = 3.34$, $p<0.002$), indicating an age difference here only for visuo-spatial ability, and no consistent differences in digit span, or number of prescription medicines.

Table 5.3: Means and Standard Deviations for Parametric Measures Confined to Older Participants

	Young-Old			Old-Old		
	Valid N	Mean	Std Deviation	Valid N	Mean	Std Deviation
Digits Forward	N=75	6.49	1.12	N=30	6.37	1.16
NPM	N=163	2.35	2.11	N=59	2.66	1.67
MCQ Total	N=79	106.49	27.56	N=31	87.42	25.33

Individual differences measures, including Everyday Activities Questionnaire (EAQ) data, were also analysed in terms of those of the older age group who had suffered any one of the serious illnesses asked about on the questionnaire (diabetes, heart or circulatory disease, stroke, high blood pressure, depression, bronchitis, liver or kidney disorders, cancer, glaucoma or cataracts, and 'other serious illness') or not. Those who indicated any one of these disorders were significantly older than those who did not ($t (220)=2.15, p<0.04$), although the mean difference was small (2.29 years). This reinforces the OO group's self reported poorer health, but more detailed comparisons for individual disorders yielded mostly non-significant differences, although those who suffered high blood pressure were older ($t (220) = 2.55, p<0.02$), and performed more poorly on AH4 Spatial ($t (103)=2.78, p<0.02$). Those who suffered Glaucoma or Cataracts were also significantly older ($t (220) =2.54, p<0.02$).

Overall these illness comparisons are both good and bad in research terms. On the good side the lack of differences with regard to cognitive and visuo-spatial measures means that within the sample these variables seem to make little difference, meaning that participants with these disorders will not have to be discounted on the grounds of illness in the experimental stage. On the negative side there is no corroboration for earlier work, although it should be noted that the sample as a whole consisted of community dwelling participants, and that those who took part in background testing and experimental work were required to travel either to the University or a local centre to take part. It seems reasonable to suppose that this sample would by definition be healthy relative to those chosen to take part in studies comparing acutely or chronically ill participants to a 'normal' population. Although this sample's cognitive ability seems relatively unaffected by illness this does not by any means constitute evidence that illness cannot be responsible for cognitive decline.

5.3: Correlational Analyses

In order to evaluate the relationships between age, background and questionnaire data a correlational analysis was performed. Variables common to all age groups entered into the initial analysis were Age, AH4 Verbal, AH4 Spatial, AH4 Total, MHV scores, Digit Cancellation, and Years of Education. Other measures included were the MacQuarrie Test for Mechanical ability and Digits Forward scores from the older groups' background assessment, and self assessed hearing and vision quality, general health, fitness, and extent of any memory decline as well as number of prescription medicines, Everyday Activity Questionnaire Necessary, Voluntary, and Total scores and the health limits measures from the questionnaire.

Age itself was negatively correlated with some measures common to all age groups: AH4 Verbal ($r= -0.157$, $p<0.003$), AH4 Spatial ($r= -0.553$, $p<0.001$), AH4 Total ($r= -0.437$, $p <0.001$), Digit Cancellation ($r= -0.509$, $p<0.001$), and Years of Education ($r= -0.395$, $p<0.001$). Age was also positively related to MHV scores ($r= 0.61$, $p<0.001$). In terms of measures completed only by YO and OO groups Age was negatively related to the MacQuarrie Test for Mechanical Ability total score ($r= -0.394$, $p<0.001$), and self rated vision ($r= -0.202$, $p<0.003$) and hearing ($r= -0.194$, $p<0.005$) quality. All other questionnaire measures were uncorrelated with age, although a negative relationship between Age and EAQ Necessary scores approached statistical significance ($r= -0.129$, $p= 0.055$).

Other than Age AH4 Total scores were, unsurprisingly, correlated with both AH4 Verbal ($r= 0.865$, $p<0.001$) and AH4 Spatial ($r= 0.9$, $p<0.001$) and AH4 Spatial and Verbal scores were also correlated ($r= 0.668$, $p<0.001$). Beyond this AH4 Verbal scores were correlated with MHV (r= 0.351, p<0.001), Digit Cancellation (r= 0.238, p<0.001), Years of Education (r= 0.257, p<0.001), MacQuarrie total (r= 0.671, p<0.001), and Digits Forward (r= 0.287, p<0.001) scores. AH4 Spatial scores were furthermore correlated with Digit Cancellation (r= 0.387, p<0.001), Years of Education (r= 0.37, p<0.001), Self Rated Vision (r= 0.206, p<0.04), and MacQuarrie total (r= 0.579, p<0.001) scores. AH4 Total correlated with MHV (r= 0.107, p<0.05), Digit Cancellation (r =0.367, p<0.001), Years of Education (r= 0.382, p<0.001), MacQuarrie total (r= 0.702, p<0.001), Digits Forward (r= 0.247, p<0.02), and EAQ Total (r= 0.193, p<0.05).

The correlation between MHV and AH4 scores probably reflect the AH4 Verbal scale's demands on vocabulary through verbal reasoning. This line of reasoning is reinforced by the weaker correlation between AH4 Total and MHV, and the lack of an association between AH4 Spatial and MHV. One may furthermore suppose that the correlation may result from more able and knowledgeable undergraduates because of the Young group's superiority in the AH4. A similar argument could explain the correlations between AH4 scores and Digit Cancellation and Years of Education: since younger people scored better on these measures anyway and had, on average, received more education it is perhaps unsurprising that they are associated. Only the older group completed Digits Forward and the association between this and AH4 Verbal may reflect the demands of manipulating numerical information. As before the lack of a relationship between this and AH4 Spatial indicates that the association between Digits Forward and AH4 Total results from the Verbal component only.

Perhaps the most surprising association is that between MacQuarrie test scores and AH4 measures. These were stronger than between AH4 and all other measures, including Age itself. The fact that younger people did not complete the MacQuarrie test militates against the explanation that the correlation merely reflects gross age differences. Furthermore it weakens this explanation in accounting for relationships between AH4 and Digit Cancellation as MacQuarrie and Digit Cancellation tests both tests involve basic perceptual and motor skills and are positively correlated ($r = 0.324$, $p < 0.07$). Indeed the correlation between AH4 and Digit Cancellation scores could be taken as evidence for a processing speed explanation although this account is weakened by the far stronger relationship between AH4 and the more complex MacQuarrie test. Even more surprisingly MacQuarrie test scores were positively associated with the untimed MHV ($r = 0.293$, $p < 0.003$) whereas Digit Cancellation was negatively associated with this measure ($r = -0.178$, $p < 0.02$). This suggests dissociation between the two measures, although quite why sensori-motor ability should be related to vocabulary in an ageing population is unclear.

Years of Education was correlated with both MacQuarrie test scores ($r = 0.371$, $p < 0.001$) and Digit Cancellation ($r = 0.269$, $p < 0.001$), although bear in mind that in the latter case this could be due to the Younger group's better performance on this measure and greater average years in education. MacQuarrie test scores were also associated with Self Rated Vision ($r = 0.199$, $p < 0.04$), Health ($r = 0.191$, $p < 0.05$), and EAQ Voluntary ($r = 0.276$, $p < 0.005$) and Total ($r = 0.286$, $p < 0.004$). Additionally Digit Cancellation was associated with Self Rated Vision ($r = 0.206$, $p < 0.04$), Health ($r = 0.253$, $p < 0.01$), and Fitness ($r = 0.217$, $p < 0.03$). It is perhaps unsurprising that measures that rely on vision such as the Digit Cancellation task and MacQuarrie test are associated with vision and this may reflect the accuracy of self-ratings in this area. The problems associated with basic perceptual and sensori-motor skills may be associated with self-ratings of general health and fitness because they may adversely affect people's perceptions of how fit and healthy they feel. Alternatively they may be symptomatic of underlying health problems and would clearly limit the extent of unnecessary activity, hence the associations with EAQ scores.

The correlation between AH4 scores and both MacQuarrie and Digit Cancellation scores may reflect either slowed processing speed, slowed perceptual motor speeds or indeed difficulty with reading the AH4 question booklets, although large print versions were made available for those who felt they needed them. Whilst this explanation may suffice for associations with Digit Cancellation ratings it cannot explain the associations between MacQuarrie scores and the untimed MHV, where participants could take as long as they wished to read and respond to the requirements of the test.

Other significant correlational relationships are detailed in Table 5.4 (below). These relationships, however, are between questionnaire items and are not directly relevant to the present research since they do not directly impinge on standardised measures of cognitive ability.

This completes the account of relationships between background and questionnaire measures. Although much of what has been described is unsurprising the associations between MacQuarrie test scores and measures of both crystallised and fluid intelligence seems somewhat paradoxical and difficult to account for. Since these associations are stronger and more broadly applicable than those between the Digit Cancellation task and other variables a simple speed of processing account may seem inappropriate in explaining general cognitive decline. Whilst this may be a compelling research question in its own right the purpose of this part of the investigation was to assess the suitability of background variables for inclusion as possible predictors of associative learning ability in multiple regression equations applied to experimental rather than correlational data.

This aim is consistent with most of the ageing literature but not with cognitive psychology broadly. Background data are important since they can tell us something about possible causes for cognitive decline with age but this principle could easily be applied to investigations concerning 'normal' cognitive ability. Surely it would be in the interests of cognitive psychology more broadly to adopt background testing in order to identify factors that have a bearing on different cognitive abilities. This would enable researchers to address questions regarding the relationships between abilities and to be more precise concerning what factors underlie a particular ability such as associative learning.

Equally background data can tell us whether our samples of participants can be considered representative of a general population. Again this would be useful information in any cognitive psychology experiment; allowing researchers to gauge more accurately how populations may differ and in what respects they are similar. Background testing is, therefore, a useful and informative exercise irrespective of whether cognitive ageing is involved, and cognitive research may benefit from a more rigorous approach.

Whilst it is important to include as many predictor variables as possible to allow a comprehensive analysis it is equally important to be parsimonious since an overabundance of variables would have inevitably reduced the power of the analysis to detect significant associations and predictions. To this end only background and questionnaire measures that correlate with Age and AH4 Total will be included, in addition to these two factors. There is, however, some controversy around which parts of the AH4 test constitute ‘fluid intelligence’.

Most researchers seem to use a summed AH4 Total score (e.g. Parkin & Java, 1999, 2000; Rabbitt et al. 1993, 2004), although others use just the verbal and numerical scale (e.g. Rabbitt, Chetwynd, & McInnes, 2003) and yet others the spatial scale (e.g. Darby & Maylor, 1998). Since AH4 Spatial and Verbal correlated highly with each other and even more so with AH4 Total the present research will not enter them into regression analyses separately and will instead use the summed scale for analyses. Both the Digit Cancellation and MacQuarrie tests will be entered since they are dissociable to an extent. Years of Education will also be used as a predictor variable, as will MHV scores.

Table 5.4: Correlations Between Questionnaire Variables

Covariate 1	Covariate 2	r	p
Years of Education	EAQ Voluntary	0.386	<0.001
Years of Education	EAQ Total	0.354	<0.001
Years of Education	Health affects Social Life	0.174	<0.01
Years of Education	Self Rated Health	0.232	<0.001
Years of Education	Self Rated Fitness	0.156	<0.03
Self Rated Vision	Self Rated Hearing	0.346	<0.001
Self Rated Vision	EAQ Necessary	0.158	<0.02
Self Rated Vision	EAQ Voluntary	0.197	<0.003
Self Rated Vision	EAQ Total	0.216	<0.01
Self Rated Vision	Self Rated Health	0.326	<0.001
Self Rated Vision	Self Rated Fitness	0.242	<0.001
Self Rated Vision	Self Rated Memory Decline	-0.26	<0.001
Self Rated Hearing	EAQ Necessary	0.155	<0.025
Self Rated Hearing	EAQ Voluntary	0.15	<0.027
Self Rated Hearing	EAQ Total	0.195	<0.005
Self Rated Hearing	Self Rated Health	0.315	<0.001
Self Rated Hearing	Self Rated Fitness	0.222	<0.002
Self Rated Hearing	Self Rated Memory Decline	-0.23	<0.002
EAQ Necessary	EAQ Voluntary	0.258	<0.001
EAQ Necessary	EAQ Total	0.453	<0.001
EAQ Voluntary	Health affects Social Life	0.315	<0.001
EAQ Voluntary	EAQ Total	0.978	<0.001
EAQ Total	Health affects Social Life	0.308	<0.001
EAQ Voluntary	Self Rated Health	0.288	<0.001
EAQ Voluntary	Self Rated Fitness	0.191	<0.004
EAQ Total	Self Rated Health	0.258	<0.001
EAQ Total	Self Rated Fitness	0.194	<0.005
Health affects Social Life	Self Rated Health	0.478	<0.001
Health affects Social Life	Self Rated Fitness	0.489	<0.001
Health affects Social Life	Self Rated Memory Decline	-0.195	<0.005
Self Rated Health	Self Rated Fitness	0.719	<0.001
Self Rated Health	Self Rated Memory Decline	-0.279	<0.001
Self Rated Fitness	Self Rated Memory Decline	-0.191	<0.005

Chapter 6: Formative Experiments I: Negative and Positive Patterning

6.1: Preface to the Experiments

As stated in the previous chapter the probability level accepted as showing a statistically significant result was $\alpha = 0.05$. Eleven experiments were conducted during the course of the studies and although designs varied, there were a number of common factors. The reader should assume, unless otherwise stated, that the following general methods were adhered to.

6.1.1: Procedures

All experiments used the same basic paradigm in the form of a human conditional learning task. Most used a food allergy problem as employed by Shanks and colleagues (Shanks, Charles, Darby, and Azmi, 1998; Shanks, Darby, & Charles, 1998; Shanks & Darby, 1998) and first developed by Wasserman (1990). Note, however, that Experiments 7 & 8 used different stimuli and responses and these will be discussed in Chapter 7. As described earlier, the food allergy task requires participants to learn which food items or combinations of items lead to an allergy, and which do not. Participants in all cases received a varying number of trial types randomised within blocks that consisted of single presentations of all stimulus-response contingencies. Two versions of each experiment were presented to different participants and the order of stimulus presentations altered to control for order effects. Stimuli were randomly drawn from a pool of food or other items. Food items had been ranked by a sample of older volunteers and undergraduates in terms of the likelihood they would cause an allergy, and consistently highly ranked foods excluded. Furthermore all stimuli were reassigned between versions such that if they predicted an outcome in one version they would predict no outcome in the other version to further account for any preconceptions concerning foods and allergies. Stimuli were presented as a Powerpoint presentation using a Hewlett Packard Omnibook 4150 laptop computer linked to a Sanyo data projector. Participants were told that the experiment would examine how difficult it was for people to learn the relationships between foods and different types of allergic reactions. They were asked to ignore any knowledge they may have about food allergies, as this would not help them during the experiment. Responses were to be made in a booklet provided and each food or foods would lead to one of several outcomes: an Allergy type or simply Allergy, or No Allergy. Participants were asked to make an outcome prediction following each presentation of food or pair of foods, and having done so they were immediately told the correct outcome for that trial. Food compounds were presented one over the other and this order of presentation was counterbalanced across trials. Each trial and its outcome were numbered and corresponded to participants' response booklets. To account for the possibility that older participants' performance may have been compromised by an inability to see stimuli and the answer book or have difficulty switching focus from one to the other both items used large font sizes. Answer sheets used a bold 20-point Times New Roman font and Powerpoint slides used a similar 40-point high contrast yellow font on a blue background. Answer booklets were also available as large print A3 size photocopies of the A4 originals if participants indicated they would need to use such an item. Afterwards participants were debriefed and given the opportunity to ask questions. The following instructions were printed in participants' answer booklets and were verbally reinforced.

Participants were encouraged to ask questions if they did not completely understand the instructions.

The learning task requires you to make a prediction about which food types cause an allergy, and which do not. You will be guessing at first but will become more accurate as the task progresses.

*This is an **artificial** categorisation task; the allergies are hypothetical and are not necessarily associated with any real food allergies.*

Simply tick the box underneath the prediction you make for each food as it is presented on the screen. Each trial is numbered and you should match the numbers on your answer sheets with the numbers on the screen.

When you have recorded each prediction look up so I know you have finished, otherwise the whole process will take much longer than necessary.

Please don't make notes or crib sheets, the task is designed to be completed unaided and to do otherwise would invalidate the results.

6.1.2: Results

Other than Experiment 1 all experimental data were dealt with in the following way prior to analysis. Participants' response booklets were collected and their responses entered into a spreadsheet as 1 for an outcome prediction and 0 for a no outcome prediction. Responses to trials resulting in no outcome (which should be 0 if correct) were then subtracted from responses to trials resulting in an outcome (that should be 1, if correct) to derive a score for each trial. This procedure derived a number between 1 and –1 that showed how far participants correctly discriminated between stimuli. For instance, had a participant correctly responded to both allergy and no allergy trials they would receive a score of 1 (i.e. 1 - 0= 1). If, however, a participant responded incorrectly to both stimuli then they would receive a score of –1 (i.e. 0 - 1= -1), and a score of 0 indicates chance levels of responding. This score therefore shows how far participants discriminated between stimuli that led to an outcome and those that led to no outcome. This method of calculation differs slightly from that employed by Shanks (Shanks, Charles, Darby, and Azmi, 1998; Shanks, Darby, & Charles, 1998; Shanks & Darby, 1998) in that the former studies tested discrimination on the basis of significant differences between Allergy and No Allergy predictions. Whilst the present studies do use this method for some analyses it was felt that calculating discrimination scores in the manner outlined above was more parsimonious for other types of analysis. These 'discrimination' scores were then treated in a number of ways to reflect different aspects of participants' performance. For the purposes of analysis discrimination scores were averaged over several trials to derive 5 blocks per experiment or stage in order to see how responses changed over the course of experiments. Thus each block in an experiment or experimental stage that presented 10 trials of each stimulus would be the average of responses to two stimulus presentations, and each block in a 15-trial experiment or stage was the average of responses to three stimulus presentations. Further analysis was also performed on overall and final trial accuracy measures. Here scores coded as 0 for an incorrect prediction and 1 for a correct response were summed over the whole experiment or stage to derive overall accuracy, whilst scores calculated in the same way for the final trial of the experiment were summed to give an indication of how accurately problems had been ultimately learned.

6.2 Negative and Positive Patterning Problems

Experiments 1 to 5 all involve negative and positive patterning problems. Recall that PPPs require participants to learn that compounds predict an outcome (in this case an allergy) whereas elements do not (e.g. A-, B-, AB+; where + indicates an Allergy outcome and - represents a No Allergy outcome). Conversely NPPs require the opposite discrimination: elements lead to outcomes but compounds don't (e.g. A+, B+, AB-). Remember too that both types of problem can be solved by adopting a rule of conjunction: elements and compounds have opposite outcomes. The following experiments varied the difficulty of the problems systematically in order to identify the influence of task complexity on learning. Furthermore participants were asked whether they had used any rules to help them complete the task in experiments 2-5. This not only allows us to identify the effects of age on learning but also the interaction of age and complexity. Because these formative studies are based on the same fundamental design and seek to address similar issues they will be grouped together in a single chapter so that an overview will be possible.

6.3: Experiment 1

Experiment 1 was a replication of Experiment 1a from Shanks, Darby, Charles and Azmi (1998) and served to begin the study by verifying whether the paper and pencil version used for the present series of studies gave results compatible with the original study. The experiment involves two concurrent NPPs and two concurrent PPPs with No Allergy and Allergies 1-4 as outcomes. There are a number of theoretical predictions that could be made concerning this experiment, particularly from quantitative theories that would help to ascertain the extent of any change in generalisation processes with age for these two types of problem.

The Rescorla-Wagner (1972) theory would predict that the PPP would be insoluble since each element would be associated with an outcome or no outcome equally frequently. Assuming a threshold of activation when $\lambda>0.5$ would, however, render the problem linearly soluble since each compound would acquire a net associative value of 1λ by summation of elements' associative weights of 0.5λ. As a consequence the compound would be associated with the presence of an allergy, whereas the elements would not be activated sufficiently to activate this association.

Unique-cue theories such as Rescorla's (1973) model would easily predict learning since, for the problem A-, B-, ABX+, the unique cue X would acquire all associative strength since it would be the only 'element' that consistently predicted the outcome, and cue-competition would ensure that elements A and B would acquire no associative value since their capacity to predict the outcome is inconsistent.

Pearce's (1987, 1994) model would, equally easily, predict that the problem be learned. A compound AB would have a similarity of 0.5 in relation to the elements A and B, and these elements would have a similarity of 0.5 to the compound, but no associative strength would be generalised between elements. For the net associative strength of V_{AB} to equal 1λ and for those of V_A and V_B to equal 0λ the associative strength of the compound AB would be 2λ and that of the elements A and B would be -1λ. This can be proved through the calculations $V_{AB} = 2 + ((0.5 \cdot -1) + (0.5 \cdot -1)$ and $V_A / V_B = -1 + (0.5 \cdot 2)$, which show that, after generalisation has been accounted for, $V_{AB} = 1$ and $V_A / V_B = 0$, allowing the model to correctly anticipate responses.

Strictly elemental theories such as the Rescorla-Wagner (1972) model would not predict learning of a NPP since each element would be associated with an outcome or no outcome equally frequently. Consequently elements would acquire associative strengths of 0.5λ and compounds would have a weight of 1λ by summation, whereas the problem requires that compounds reach an associative weight of 0λ, and elements a value of 1λ. In essence the model is incapable of discriminating between NPPs and PPPs.

Rescorla's (1973) unique cue model could solve the problem A+, B+, ABX- by assuming that the unique cue X would acquire an associative strength of -2λ in order to counteract the associative strengths accrued by the elements A and B of 1λ, although this would require the further assumption that unique cues could acquire associative strengths greater than λ.

Pearce's (1987, 1994) model makes the uncontroversial prediction that the NPP would be learned if compound AB acquired an associative strength of –2λ and the elements A and B values of 2λ since $V_{AB} = -2 \cdot ((0.5 \cdot 2) + (0.5 \cdot 2))$, $V_A / V_B = 2 + (0.5 \cdot -2)$ and therefore, after generalisation, $V_{AB} = 0$ and $V_A / V_B = 1$. Note that both the unique-cue and Pearce's model suggest that more learning is necessary to solve a NPP and therefore predict that such problems should take longer to learn and engage more processing resources, and therefore participants' ability to learn NPPs should show a greater age related decline than learning of PPPs.

Individual differences variables will also be entered into multiple regression analyses using the enter method to see if Age, AH4 Total, Digit

Cancellation (DC), MacQuarrie test of mechanical ability (McQ), Years of Education (YE) and Mill Hill Vocabulary scale (MHV) measures are able to predict learning accuracy. Since all these factors may, theoretically, mediate learning no predictions are offered at this stage concerning the nature and extent of their influence on learning and generalisation processes.

6.3.1: Participants

Older participants were 25 volunteers from the Wolverhampton Ageing and Memory Project. Their ages ranged from 61 to 83 with a mean of 72.57 (S.D. = 6.14). The Young Old (YO) group consisted of thirteen participants with a mean age of 67.76 (S.D. = 4.1), and the Old Old (OO) group had twelve members with a mean age of 77.7 (S.D. = 2.67). Younger participants were 12 undergraduate volunteers, their ages ranged from 19 to 32 with a mean of 22.51 (S.D. = 4.73).

Table 6.1: Participant Summary Statistics

	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Age	22.51	4.73	67.76	4.10	77.70	2.67
Years of Education	14.00	.00	11.38	2.06	11.67	2.77
AH4 Total	87.73	18.34	61.08	24.03	67.63	14.25
Digit Cancellation	24.36	5.14	15.62	2.60	15.00	4.41
Mill Hill Vocabulary Scale	27.18	4.31	32.70	4.30	29.36	10.93
MacQuarrie Total	.	.	95.20	25.67	93.80	20.15

Table 6.2: Experiment 1 Design and Stimuli

Abstract Form	*Foods Version 1*	*Foods Version 2*	*Outcome*
A	Cheese	Fish	Allergy 1
B	Chocolate	Banana	Allergy 1
AB	Cheese & Chocolate	Fish & Banana	No Allergy
C	Milk	Olive Oil	Allergy 2
D	Eggs	Avocado	Allergy 2
CD	Milk & Eggs	Olive Oil & Avocado	No Allergy
E	Fish	Cheese	No Allergy
F	Banana	Chocolate	No Allergy
EF	Fish & Banana	Cheese & Chocolate	Allergy 3
G	Olive Oil	Milk	No Allergy
H	Avocado	Eggs	No Allergy
GH	Olive Oil & Avocado	Milk & Eggs	Allergy 4

6.3.2: Design & Materials

There were two NPPs and two PPPs presented concurrently in the form of a food allergy task, as detailed earlier. The experiment was a mixed design with age group as the between subjects factor (old and young), and trials (1-10), stimuli (elements and compounds), and problem (negative and positive patterning problems) as within subjects factors. Table 6.2 shows the experimental design. Foods A, B, C, D, and the compounds AB and CD constituted the negative patterning problem whilst foods E, F, G, H, and the compounds EF and GH comprised the positive patterning problems. There were five possible outcomes: allergies 1-4 and No Allergy. Participants received ten trials of each type randomised within blocks of twelve stimulus types, making 120 trials in total.

6.3.3: Procedure

The standardised procedures outlined above were adhered to in this experiment, other than participants were informed that there were five possible responses to each stimulus: No Allergy and Allergies 1-4.

6.3.4: Results: Initial Analysis

The initial analysis was by a mixed 3 (Age) by 2 (NPP or PPP: Problem) by 5 (Blocks of 2 trials) ANOVA. Data were initially entered as 1 for a correct prediction and 0 for an incorrect prediction. These scores were then adjusted on No Allergy trials by subtracting 1 from coded scores so that correct No Allergy responses were worth 0 and Incorrect No Allergy responses 1. After that discrimination scores were calculated for each stimulus and problem type over five blocks of two trials, and raw scores used to calculate final trial and overall accuracy.

There were no significant differences overall between age groups ($F_{(2,34)} = 1.48$, $p>0.05$) in this experiment. There was a significant effect of Blocks ($F_{(4,136)} = 44.01$, $p<0.001$), showing simply that participant responses changed as the experiment progressed. Figure 6.1 and Table 6.3 suggests that this change reflected increasing accuracy.

Figure 6.1: Age Group by Blocks

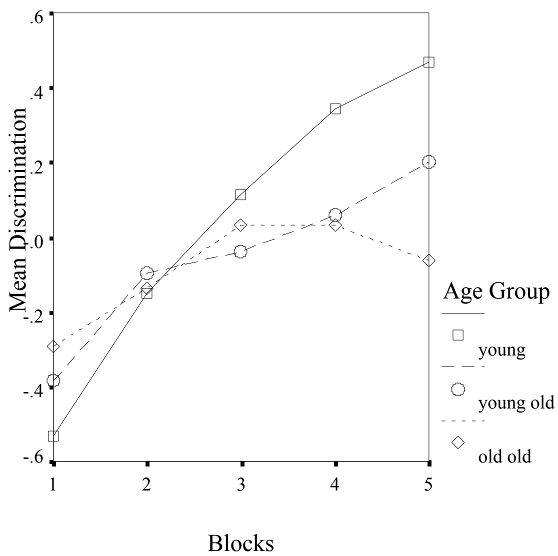


Table 6.3: Summary Statistics for Figure 6.1

	Young		Young Old		Old Old	
Block	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	-.53	.12	-.38	.21	-.29	.19
2	-.15	.16	-.10	.28	-.14	.28
3	.11	.28	-.04	.30	.03	.31
4	.34	.29	.06	.31	.03	.31
5	.47	.38	.20	.32	-.06	.35

The Age by Blocks interaction (see Figure 6.1) was also significant ($F_{(8,136)} = 5.9$, $p<0.001$), showing that the Y group's responses changed more and became more accurate than the YO and OO groups' as the experiment progressed. This interaction was not significantly attenuated by AH4 Total scores when these were entered as a covariate in an ANCOVA ($F_{(8, 124)} = 2.62$, $p<0.012$) This suggests that age itself may be a greater arbiter of speed of learning than fluid intelligence in this experiment. There was also a significant interaction between Problem and Blocks ($F_{(4,136)} = 3.34$, $p<0.013$; see Figure 6.2), reflecting the overall superiority of participants at solving the PPP over the NPP. Although this trend was more marked in the older age groups the Problem by Age by Blocks interaction was non-significant, and the addition of AH4 as a covariate rendered this interaction non significant ($F_{(4,124)} = 0.39$, $p>0.05$), suggesting that any differences in learning problems may originate in differences in fluid intelligence regardless of age.

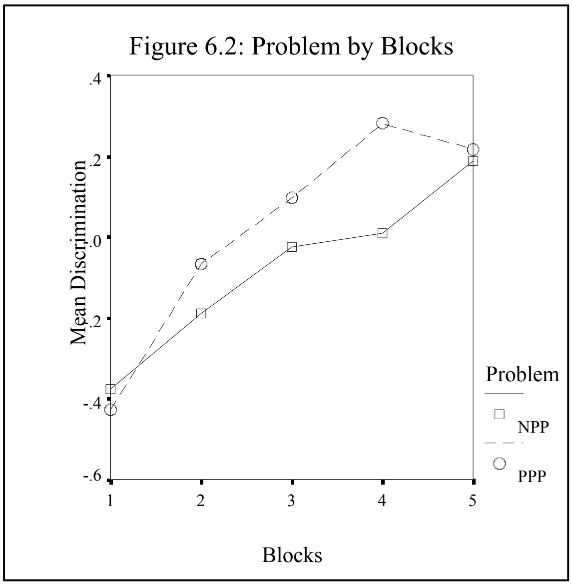


Table 6.4: Summary Statistics for Figure 6.2

	PPP		NPP	
Block	Mean	Std. Deviation	Mean	Std. Deviation
1	-.43	.26	-.38	.31
2	-.07	.26	-.19	.36
3	.09	.28	-.03	.46
4	.28	.33	.01	.51
5	.22	.38	.19	.54

Further analysis was conducted on participants' responses on the final trial to indicate how well they had learned the problems overall. Age differences in overall accuracy on the final trial were tested by a one way ANOVA. This showed that older people's accuracy was significantly worse on the final trial ($F_{(2,34)} = 3.9$, $p<0.03$). A Bonferroni post-hoc test showed that the difference was confined to that between Y and OO groups (Mean Difference = 0.25, $p<.03$). Interestingly, this age difference was attenuated by AH4 scores ($F_{(2,31)} = 0.243$, $p>0.05$) when this variable was entered as a covariate. For each age group t-tests were then performed to compare their final trial outcome predictions on elements and compounds on each problem. A significant difference shows participants were consistently discriminating between outcomes for elements and compounds on the final trial and therefore whether they had ultimately learned the problems. These revealed that the Y group had learned both the PPP ($t=3.08$, $df= 11$, $p<0.02$) and the NPP ($t=3.84$, $df = 11$, $p<0.004$). The YO group learned the PPP ($t=2.35$, $df= 12$, $p<0.04$), but not the NPP ($t=0.501$, $df= 12$, $p>0.05$), and the OO group had learned neither problem (PPP: $t=0.17$, $df= 11$, $p>0.05$; NPP: $t=0.11$, $df= 11$, $p>0.05$). One-Way ANOVAs were then performed separately for each problem to see if final trial discriminations for NPPs and PPPs differed with age (see Figure 6.3, overleaf). This revealed a difference between age groups for NPPs ($F_{(2,34)}=3.75$, $p<0.035$), but not PPPs ($F_{(2,34)}=2.31$, $p>0.05$). Bonferroni post-hoc tests showed that the Y group were significantly more accurate at solving the NPP than the OO group (Mean Difference = 0.604, $p<.05$) and that this difference was confined to these two groups. This demonstrates that older participants ultimately learned the discriminations less well than younger participants and that the linear insolubility of the NPP makes it practically, as well as theoretically more difficult than the PPP for older people. Again, though, Age differences here were attenuated by the introduction of AH4 as a covariate ($F_{(2,31)} = 0.325$, $p>0.05$), suggesting that a general fluid intelligence factor is more important in predicting learning difficult problems than age as such.

Figure 6.3: Final Trial Discrimination by Problem

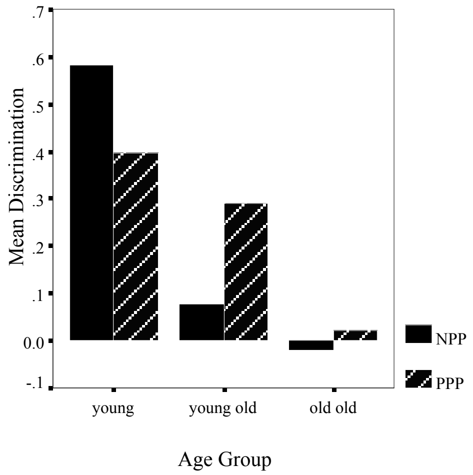


Table 6.5: Summary Statistics for Figure 6.3

Problem	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
NPP	.58	.53	.08	.55	-.02	.66
PPP	.40	.45	.29	.44	.02	.43

Figure 6.4: Final Trial Accuracy by Stimulus

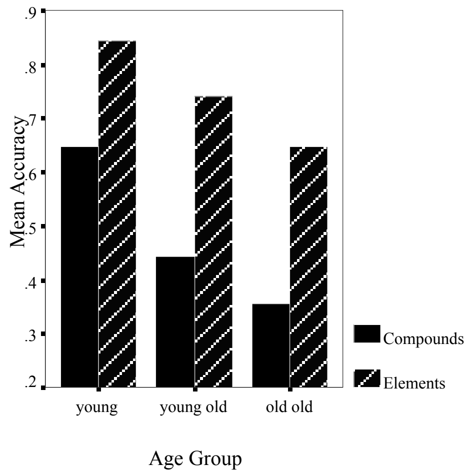


Table 6.6: Summary Statistics for Figure 6.4

Stimulus Type	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Compound	.65	.25	.44	.25	.35	.25
Element	.84	.24	.74	.26	.65	.27

A second analysis was conducted to test for differences in learning contingencies involving elements and compounds. This was initially achieved through an Age (3) by Blocks (5) by Stimulus (2: elements and compounds) mixed ANOVA. This revealed main effects of Stimulus ($F_{(1,34)}$ =44.02, p<0.001) and Blocks ($F_{(4, 136)}$ =44.01, p<0.001), showing that participants' discriminations improved over the course of the experiment and that discriminations were significantly more accurate for elements than for compounds. Furthermore the Age by Blocks ($F_{(8,136)}$ =5.9, p<0.001) and Stimulus by Blocks ($F_{(4,136)}$ =2.84, p<0.03) interactions were significant, suggesting that younger people learned the problems more quickly and that all participants' accuracy improved more quickly for elemental stimuli than compound stimuli.

Beyond this age group comparisons were made with separate one way ANOVAs for final trial accuracy on elements and compounds. They revealed a significant effect of age on compound ($F_{(2,34)}$ =4.29, p<0.03) but not element final trial accuracy ($F_{(2,34)}$ =1.79, p>0.05: see Figure 6.4). A Bonferroni post-hoc test demonstrated that once more the age difference was solely between Y and OO groups (Mean Difference = 0.292, p<.03). Again, entering AH4 as a covariate eliminated the age differences in compound learning accuracy ($F_{(2,31)}$ = 0.65, p>0.05).

6.3.5: Results: Multiple Regression

To decide which of the variables decided on in Chapter 5 to enter into a multiple regression analysis using the enter method an initial correlation was performed between potential predictor variables (Age, Years of Education (YE), Digit Cancellation (DC), MacQuarrie Total (McQ), MHV, and AH4 Total) and dependent variables (overall (OA) and final trial (FTA) accuracy). This analysis revealed significant associations between FTA and Age (r= -0.481, p<0.04), YE (r = 0.573, p<0.001), DC (r = 0.355, p<0.05), and AH4 (r = 0.517, p<0.003). Similarly OA was correlated with Age (r = -0.347, p<0.04), YE (r = 0.559, p<

0.001), DC ($r = 0.360$, $p<0.05$), and AH4 ($r = 0.386$, $p<0.03$). Neither FTA nor OA were associated with McQ or MHV scores and consequently these variables were not entered as predictors of either OA or FTA in the subsequent multiple regression analysis.

In terms of OA the multiple regression model using the enter method was significant ($F_{(4,27)} = 3.25$, $p<0.03$, Adjusted $R^2 = 0.225$), indicating that overall the predictor variables accounted for a little under a quarter of the variability in OA. The only significant predictor variable to emerge, however, was YE (Beta = 0.476, $p<0.03$), whereas all other predictor variables were non-significant.

The regression model was also a significant predictor of FTA ($F_{(4,27)} = 4.89$, $p<0.04$, Adjusted $R^2 = 0.334$). Although the overall model accounted for more of the variance in FTA none of the individual predictor variables were significant, although YE approached significance (Beta = 0.375, $p=0.057$).

Overall this seems to suggest that both OA and FTA could, to an extent, be predicted by the selected predictor variables. The only significant overall predictor of OA was YE, whereas FTA had no significant individual predictor variables. This analysis obviously begs the question of why YE should predict overall accuracy in a contingency learning problem.

6.3.6: Discussion

The lack of overall age differences in this experiment suggests floor effects since the Young group's final block discriminations were only 0.47, implying that this group were correctly discriminating between stimuli less than half the time during the final trial block. There were, however, some age differences in terms of an Age by Blocks interaction, and an inspection of Figure 6.1 suggests that the Y and YO groups' learning had not reached asymptote after ten trials and may have improved further, whereas the OO group's learning peaked in block 3 and became worse thereafter and, as a consequence, less likely to improve. Taken together these observations imply that the problem may have been too difficult for participants to learn and that this may have minimised any overall age differences.

Having said that, it was clear that the OO group was especially disadvantaged in that they really did not learn either NPPs or PPPs at all, suggesting an overall decline in learning consistent with the notion that older people's learning was more 'elemental' than younger participants' learning. This could be explained in terms of an overgeneralisation between stimuli. Younger people use configural strategies and can therefore learn both problems, whereas older people may use a rule of summation that leads to overgeneralisation, especially the Old-Old. Confirmation that the NPP was more difficult than the PPP came in terms of a Problem by Blocks interaction and the observation that the YO group's discriminations between elements and compounds were significantly different for the latter but not the former. This observation is also consistent with the assumption that older people should process stimuli in an elemental manner, since the OO group failed to learn either problem, as predicted by Rescorla-Wagner (1972) and the YO group only learned the PPP, elementally soluble if one adopts a threshold of activation rule, but not the NPP, which remains insoluble to any elemental theory. This may illustrate the relatively preserved capacity of the YO to use strategies that require more processing resources. Figure 6.3 illustrated a contradictory observation though; the Y group's responses on the final trial were more accurate for the NPP than the PPP. Perhaps this underlies the significant difference between YO and OO for the NPP and the lack of any differences for the PPP. One explanation could be that greater exposure to the multiple allergy contingencies in the former problem facilitated learning since there are twice as many element trials as compound trials. It is possible that greater exposure to these contingencies facilitated learning relative to the PPP, although it would be equally plausible to assert that since there were more no allergy trials in the latter problem this should have been easier too. The fact that multiple allergy outcomes existed seems to have made the problems unduly difficult and rendered any meaningful comparison between them difficult, and this will have to be addressed in subsequent experiments by using only Allergy and No Allergy response types.

The relevance of the differences between compound and element learning may not, however, have been obscured by this contamination since there were equal numbers of element and compound trials with equal numbers of Allergy and No Allergy responses. Here the OO group was significantly disadvantaged relative to the Y group in terms of compound, but not element learning. There are two possible interpretations of this observation. Firstly it seems reasonable to suppose that learning both stimulus-stimulus as well as stimulus-response associations makes compound learning especially difficult for the over 75s. This is certainly consistent with the associative deficit hypothesis (Naveh-Benjamin, 2000; Naveh-Benjamin et al. 2002; Naveh-Benjamin et al. 2003; Naveh-Benjamin et al. 2004) and an underlying MTL dysfunction. On the other hand, this could be the result of overgeneralisation between stimuli rather than an inability to form compounds. Using an elemental strategy would also obviate the need to learn about compounds since this strategy requires the assumption that responses to compound stimuli are merely the product of the associative strengths of their constituent elements. More sophisticated elemental explanations such as Rescorla's (1973) unique-cue model presumably tap into executive processing resources, which would be susceptible to age related decline. Similarly configural generalisation processes may require more strategic processing than simple elemental assumptions and be similarly subject to age related decline. This could also be explicable in the context of the observation that entering AH4 scores as a covariate attenuated age differences between problem and stimulus type. It could be that engaging configural processing resources may be more cognitively demanding than elemental strategies, and consequently tap into general intellectual abilities that decline with age as opposed to lower level learning abilities.

In terms of the multiple regression analysis it seems curious that YE should predict overall accuracy in an associative learning experiment. One interpretation could be that, as with Hebb's (1949; see Chapter 2) rats, an enriched intellectual environment led to greater complexity within the brain and increased problem solving abilities. Even allowing for the ravages of age the cognitive abilities of people with an enriched mental life through education and a subsequent intellectually demanding career should still be well preserved relative to others of their cohort. Whilst this is entirely plausible it is equally possible that this merely reflects the fact that the Young group was composed entirely of first year undergraduates who had a greater mean number of years of education and were better than the older groups at solving the problem. Despite this Age remained non-significant as an individual predictor of OA and FTA, which militates against the latter interpretation, so it seems likely to reflect the performance of both younger people and better-educated older participants.

Furthermore, the inability of Age to predict learning may be due to floor effects. Certainly, it suggests that this design was difficult for participants of all ages.

6.4: Experiment 2

To test this assumption that floor effects weakened age differences Experiment 2 was a replication of Experiment 1 with the sole difference that responses were confined to simply Allergy or No Allergy. This manipulation should not only make the problems simpler to solve but also make comparisons between problems easier since multiple responses rendered the reasons why one problem should be easier than another equivocal. Theoretical predictions remain the same, although age differences may be more marked since the decrease in complexity should allow the Y group to learn the problem better. Participants were also asked whether they had induced any rules to aid them in this experiment, and it should be expected that older participants would be less likely to induce a rule because of their slower learning and tendency to overgeneralise between stimuli.

6.4.1: Participants

Participants were 30 undergraduates (Mean Age = 24.11, S.D. = 6.57), 19 Young-Old (Mean Age = 68.09, S.D. = 3.63), and 13 Old-Old (Mean Age = 77.88, S.D. = 3.14) people.

Table 6.7: Participant Summary Statistics

	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Age	24.11	6.57	68.09	3.63	77.88	3.14
Years of Education	14.00	.00	11.84	2.54	12.46	2.63
AH4 Total	78.38	17.55	66.11	16.10	64.33	16.71
Digit Cancellation	24.27	5.77	15.89	2.88	14.25	4.97
MHV	26.54	3.82	33.50	4.03	34.45	6.23
MacQuarrie Total	.	.	100.21	28.17	98.36	20.62

6.4.2: Design & Materials

The design of Experiment 2 was, as stated, only different to that of Experiment 1 in that responses were solely Allergy or No Allergy.

6.4.3: Procedures

Procedures were as per the general procedures, outlined earlier in this chapter. One addition to the general procedures was that participants were asked if they had spotted a rule that had helped them to solve the problem and what, if any, that rule was. This was in response to the observations of Shanks and Darby (1998) that induction of a rule of conjunction for PPPs and NPPs was mediated by associative learning accuracy.

6.4.4: Results: Initial Analysis

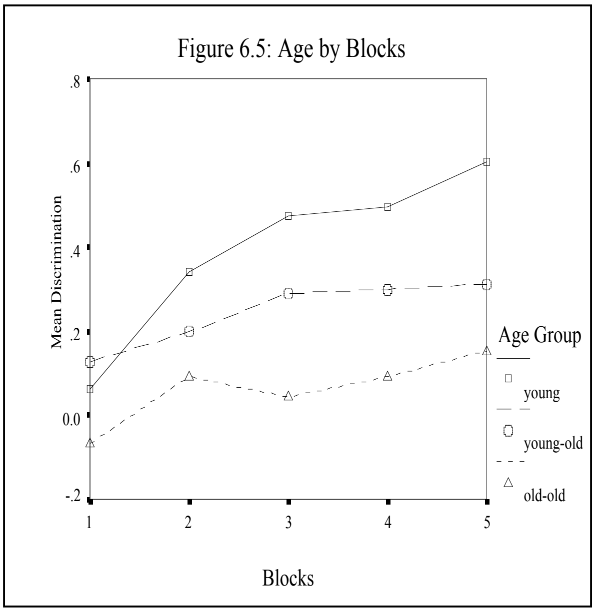


Table 6.8: Summary Statistics for Figure 6.5

	Young		Young Old		Old Old	
Block	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.06	.23	.13	.24	-.07	.26
2	.34	.24	.20	.36	.09	.38
3	.48	.23	.29	.33	.04	.31
4	.49	.30	.30	.38	.09	.29
5	.60	.35	.31	.46	.15	.35

Again the initial analysis was by a mixed 3 (Age) by 2 (NPP or PPP: Problem) by 5 (Blocks of 2 trials) ANOVA. Calculations of the discriminations for analyses were as per the procedures described earlier in this chapter.

This analysis revealed an overall effect of Age ($F_{(2,59)}=7.97$, $p=0.001$) and a Bonferroni post-hoc demonstrated that this difference was confined to that of the Y and OO groups (Mean Difference = 0.33, $p=0.001$). This age difference also remained significant after AH4 scores were entered as covariates ($F_{(2,56)}= 6.21$, $p=0.005$). There was also a main effect of Blocks ($F_{(4,236)}=19.899$, $p<0.001$), mundanely showing that participants' responses had changed over the course of the experiment, although there was no main effect of Problem, suggesting no overall difficulties with NPPs relative to PPPs. A Blocks by Age interaction (see Figure 6.5; $F_{(8,236)}=3.667$, $p=0.001$) showed that Younger participants had learned the problems more quickly than older groups, and this remained significant after entering AH4 as a covariate ($F_{(4,224)}= 5.198$, $p<0.01$).

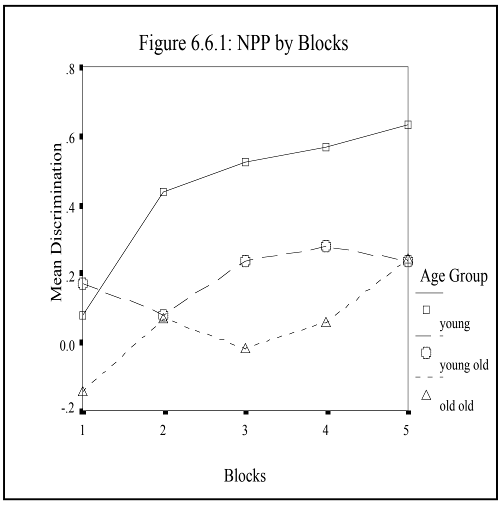
Interestingly there was a three-way interaction between Age, Problem, and Blocks ($F_{(8,236)}=2.1$, $p<0.04$: see Figures 6.6.1 & 6.6.2). This interaction is difficult to interpret but it seems that different age groups are responding in different ways to the different problems. Here Young participants learned the NPP better than the PPP again, although clearly one cannot attribute this to multiple allergy outcomes. The YO learned the PPP better than the NPP whilst the OO had learned the NPP slightly better by the end of the experiment after they had been making more accurate responses to the PPP midway through the experiment. The Young's performance on the PPP was still, however, better than the YO group's despite the fact that the YO were better at this problem than the NPP. This interaction was, however, attenuated by the entry of AH4 as a covariate ($F_{(4,224)} = 0.218$, $p>0.05$). The lack of any significant Age by Problem interaction reflects the lack of consistency around how different age groups solved NPPs and PPPs.

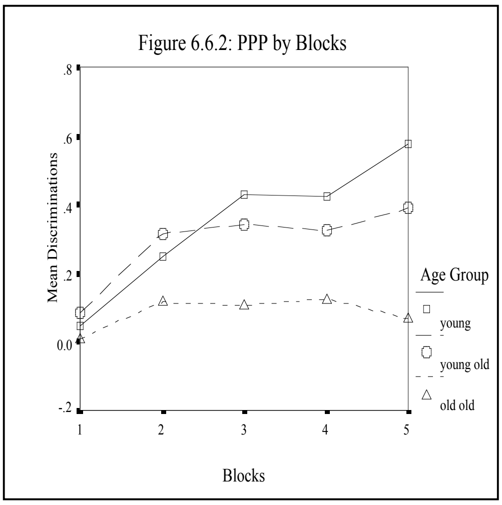
Table 6.9: Summary Statistics for Figure 6.6.1 (NPP)

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.08	.31	.17	.34	-.14	.30
2	.44	.32	.08	.53	.07	.34
3	.52	.29	.24	.38	-.02	.29
4	.57	.37	.28	.47	.06	.31
5	.63	.44	.24	.47	.24	.36

Table 6.10: Summary Statistics for Figure 6.6.2 (PPP)

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.05	.37	.09	.32	.01	.37
2	.25	.35	.32	.42	.12	.50
3	.43	.38	.34	.49	.11	.43
4	.42	.38	.32	.55	.13	.40
5	.57	.35	.39	.58	.07	.50





To ascertain whether there were any differences between response accuracies for elements and compounds a 3 (Age) by 5 (Blocks) by 2 (Stimulus) ANOVA was performed. Unsurprisingly, and identically to the previous analysis, there were main effects of Blocks ($F_{(4,236)} = 19.899$, $p<0.001$), Age (F

$_{(2,59)}$ =7.97, $p<0.001$), and Blocks and Age ($F_{(8,236)}$ =3.67, $p<0.001$). All other effects were non-significant, implying that there were no real differences here in the way participants learned compounds and elements.

As in Experiment 1 paired sample t-tests were performed to see whether participants' responses to trials resulting in allergy and no allergy were consistently different in the final block of trials. Since there were no differences between age groups in terms of stimuli or problem there were no separate analyses in terms of these factors. Here the Young's responses to trials resulting in Allergy or No Allergy were significantly different (t =7.92, df=29, $p<0.001$), as were the YO's (t =3.22, df=18, p=0.005), although the Young's mean difference (6.125) was nearly twice that of the YO's (3.55). Contrarily the OO's responses on the final trial did not differ between stimuli resulting in Allergy and No Allergy (t =1.35, df=12, $p>0.05$), indicating that they were the only group that did not learn the problems, although the means suggest that the Young learned the problem better than the YO.

6.4.5: Rule Induction

Recall that participants had been asked whether they had induced any rules to help them solve the problems. Here their responses were deemed correct if they specified a general rule of conjunction: elements and their compounds had opposite outcomes. Only six participants were correct and the remaining 56 specified either no rule or an incorrect one. All of the Rule Correct (RC) group were in the Young age group (Mean Age = 21.64, SD = 1.6) whereas the Rule Incorrect group (RI) were from all age groups (Mean Age = 51.78, SD = 24.5). There were significant differences between RI and RC groups in AH4 Total (t = 3.21, df = 55, $p<0.005$), and Digit Cancellation (DC; t = 2.84, df = 55, $p<0.007$), suggesting that these variables may be important contributors to learning and rule induction, as well as age. Although it is likely that these differences reflect the fact that the RC group were all in the Young age group it is also possible that rule learners represented the more able of their age cohort. There is partial support for this notion in that when only the Young group were entered into a comparison between RC and RI groups in terms of AH4 Total scores a significant difference emerged ($t = 2.32$, $df = 24$, $p<0.03$) although this did not extend to DC scores($t = 0.92$, $df = 24$, $p>0.05$). This suggests that general intelligence may be a bigger factor in rule induction than processing speed.

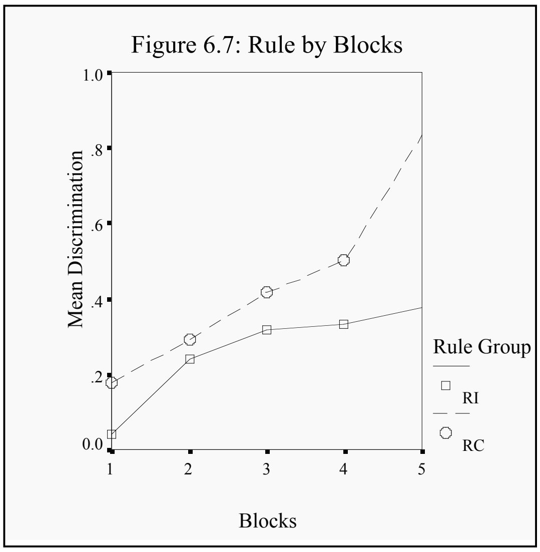


Table 6.11: Summary Statistics for Figure 6.7				
	Rule Incorrect		Rule Correct	
	Mean	Std Deviation	Mean	Std Deviation
1	.04	.24	.18	.26
2	.24	.33	.29	.22
3	.32	.33	.42	.24
4	.33	.36	.50	.33
5	.38	.42	.83	.24

Further analysis was by two ANOVAs; one was Rule (2: RC & RI) by Problem by Blocks in terms of discrimination scores and the other Rule (2) by

Stimulus (2) by Blocks (5) for accuracy scores. None of the interactions between Stimulus or Problem were significant whereas both analyses revealed identical main effects of Blocks ($F_{(4,240)}$ =17.28, p<0.001) and a Rule by Blocks interaction ($F_{(4,240)}$ =3.27, p<0.015). Figure 6.7 shows that the learning of the RI group proceeds in a classic negatively accelerated 'learning curve' and appears to reach asymptote by Block 5 with a mean discrimination of 0.38 (SD = 0.42), although clearly this group were not homogenous. The RC group, on the other hand, showed a similar but more accurate pattern until Block 4, whereupon their discrimination increases rapidly to reach 0.83 (SD =0.24) by Block 5. This suggests that rule induction happened in Block 5 and the sharp increase in discrimination accuracy reflects a 'moment of revelation' for RC participants. There was also a clear difference between groups in terms of accuracy in the final trial (t = 2.27, df = 60, p<0.02), whereas the comparison in terms of overall accuracy only approached significance (t = 1.52, df = 60, p = 0.068). This again suggests that participants in the RC group may have induced a rule relatively late in the experiment.

6.4.6: Multiple Regression

Again an initial correlation was performed between potential predictor variables (Age, Years of Education (YE), Digit Cancellation (DC), MacQuarrie Total (McQ), MHV, and AH4 Total) and dependent variables (overall (OA) and final trial (FTA) accuracy). There were significant correlations between OA and Age (r = -0.426, p<0.001), and DC (r = 0.354, p<0.008), and between FTA and Age (r = -0.442, p<0.001) and DC (r = 0.308, p<0.03). Age and DC were therefore entered as predictor variables in a multiple regression analysis using the enter method. The model proved significant for OA ($F_{(2,54)}$ =6.66, p<0.004, adjusted $R^2 = 0.168$) with Age emerging as the only significant single predictor of OA (beta = -0.381, $p<0.04$). The results for FTA were similar. The model again proved significant ($F_{(2,54)}=5.85$, $p<0.006$, adjusted $R^2 = 0.148$) with Age again emerging as the only significant predictor of FTA (beta = -0.41, $p<0.03$).

6.4.7: Discussion

In this experiment differences between age groups were significant overall, although mainly confined to differences between Y and OO groups. This suggests that the use of simple Allergy versus No Allergy outcomes had simplified the problem to the extent that younger participants could learn the problems and that this could be viewed as decreasing the complexity and demands of the problem. Furthermore, Age emerged as a strong predictor of both final trial and overall accuracy, confirming the importance of Age as a predictor over and above other individual differences factors. This suggests that conditional learning may be dissociable from other cognitive abilities, particularly general intelligence, sensori motor and perceptual speed, and this interpretation is underlined by the observation that age differences remained significant when AH4 scores were entered as a covariate. Unlike Experiment 1, however, there were no differences in terms of Problem apart from a three way interaction between Age, Problem and Blocks implying that there were few differences in the way participants learned NPPs and PPPs although younger participants learned the former more quickly. This ability to learn NPPs more quickly may also implicate higher level processing abilities, since AH4 scores rendered this interaction non-significant when entered as a covariate. This, therefore, does not rule out the assumption that older people process stimuli in a more elemental manner since there were overall age differences reflecting the relative difficulty of what are both non-linear problems. There were also no differences between element and compound accuracy, which creates difficulties for a MTL deficit interpretation of age related decline in associative learning. The observations concerning rule induction seem to confirm Shanks and Darby's (1998) assertion that rules are only induced once sufficient learning has occurred since there were highly significant differences between RC and RI groups not only in learning accuracy but also in terms of AH4 and DC scores and Age. Certainly, this experiment confirmed two things. Firstly that there are age related declines in the ability to learn HCL problems and secondly that the food allergy paradigm supports learning in a paper and pencil version. One major problem with the current experiment was, however, that there were no older participants in the RC group. This makes it difficult to say whether the observed individual differences in AH4 and DC scores between RC and RI groups were the result of ageing or whether they underlie rule induction. The next experiment should be made simple enough to allow older people to induce rules, if they are able to at all.

6.5: Experiment 3

Experiment 3 was a direct replication of Experiment 2, the only differences being that participants learned only one negative and one positive patterning problem concurrently and the number of trials was increased to 15. This reduction in problem complexity should also reduce age differences in overall performance and the ability of age to predict learning accuracy both overall and on the final trial. It will also allow the older participants more of a chance to induce rules since no participant in the YO or OO groups had done so in the previous experiment.

6.5.1: Participants

Participants in the older group were 25 volunteers, their ages ranged from 56 to 85 with a mean of 70.24 (S.D. 6.71). Young Old participants had a mean age of 67.91 (n=18; SD=5.23) and Old Old participants had a mean age of 78.2 (n=8; SD=3.89) Younger participants were 20 undergraduates who volunteered to participate in the study aged between 18 and 47 with a mean of 25.78 (SD=7.22).

Table 6.12: Participant Summary Statistics

	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Age	25.78	7.22	67.91	5.23	78.20	3.89
Years of Education	14.00	.00	13.94	3.11	12.75	2.25
AH4 Total	86.41	17.50	82.31	17.29	65.43	20.49
Digit Cancellation	22.94	6.34	17.23	3.88	16.86	3.72
MHV	27.00	4.00	34.67	3.73	34.29	1.98
MacQuarrie Total	.	.	120.08	21.78	89.43	23.87

6.5.2: Design & Materials

Table 6.13: Experimental Design

Abstract	Version 1	Version2	Allergy
A	Soup	Chicken	No Allergy
B	Lettuce	Jam	No Allergy
C	Chicken	Soup	Allergy
D	Jam	Lettuce	Allergy
AB	Soup Lettuce	Chicken Jam	Allergy
CD	Chicken Jam	Soup Lettuce	No Allergy

The experiment was a mixed design with age group as the between subjects factor (old and young), and trial blocks (1-5), stimuli (elements and compounds), and problem (negative and positive patterning problems) as within subjects factors. Table 6.13 shows the experimental design, which consists of one negative and one positive patterning problems presented concurrently. Foods A, B, and the compound AB constituted the positive patterning problem whilst foods C, D, and the compound CD comprised the negative patterning problems. There were two possible outcomes: Allergy and No Allergy. Participants received fifteen blocks of 6 trials randomised within blocks, making 90 trials in total. As can be seen in Table 6.5, foods were reassigned between versions.

6.5.3: Procedures

Procedures were as detailed in the general procedures, outlined earlier in this chapter. As in Experiment 2 participants were asked if they had spotted a rule that had helped them to solve the problem and what, if any, that rule was.

6.5.4: Results: Initial Analysis

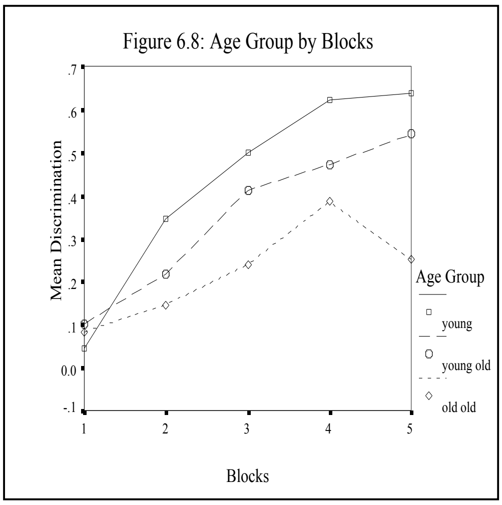


Table 6.14: Summary Statistics for Figure 6.8

	Young		Young Old		Old Old	
Blocks	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.05	.24	.10	.29	.08	.15
2	.35	.34	.22	.27	.15	.31
3	.50	.35	.41	.33	.24	.27
4	.62	.40	.47	.42	.39	.33
5	.64	.44	.54	.37	.25	.39

Initial analysis was by an Age (3) by Problem (2) by Blocks (5) mixed ANOVA with Age Group as the between subjects factor with discriminations averaged over five three trial blocks as the dependent variable. This revealed no significant effects of Age Group ($F_{(2,43)}$=1.67, p>0.05). The only significant main effects were, mundanely enough, that of Blocks (see Figure 6.8: $F_{(4,172)}$=23.14, p<0.001), and more interestingly of Problem (see Figure 6.9: $F_{(1,172)}$ = 9.34, p<0.005). Even more intriguingly the Age by Problem interaction was also significant ($F_{(2,172)}$ = 6.98, p<0.003), and remained so once the covariance with AH4 had been accounted for ($F_{(2,164)}$ = 3.38, p<0.05). These results seem to reflect an overall disadvantage at the NPP, with the OO group particularly poor at this class of problem relative to PPP, which was solved reasonably well by all groups. This implies that the oldest participants were particularly compromised when solving the NPP.

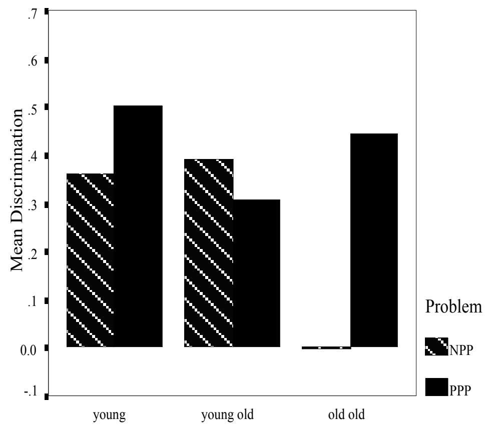


Figure 6.9: Problem by Age Group Interaction

Table 6.15: Summary Statistics for Figure 6.9

	Young		Young Old		Old Old	
Problem	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
NPP	.36	.34	.39	.26	.00	.43
PPP	.50	.31	.31	.38	.45	.17

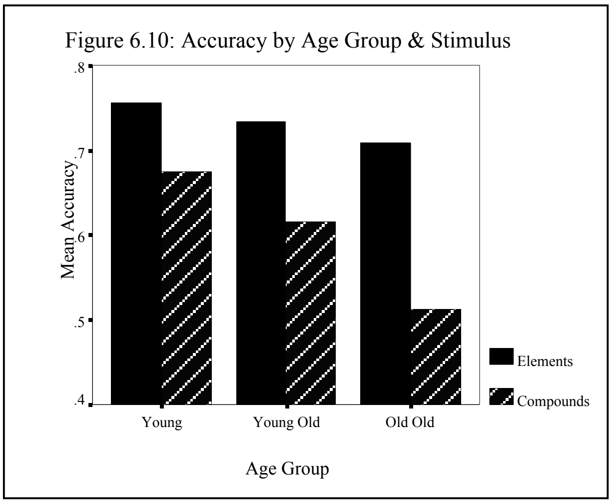


Table 6.16: Summary Statistics for Figure 6.10

	Young		Young Old		Old Old	
Stimulus	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Elements	.76	.14	.73	.16	.71	.13
Compounds	.68	.17	.61	.18	.51	.14

As in the previous experiment an Age by Blocks by Stimulus analysis was performed on response accuracies for elements and compounds over the course of the experiment. There was, again and dully enough, an identical effect of Blocks ($F_{(4,172)}=23.14$, $p<0.001$) and a non-significant Age main effect. Fascinatingly, though, in this experiment the main effect of stimulus was significant (see Figure 6.10: $F_{(1,43)}=26.62$, $p<0.001$) although there were no interactions between Age and any other variables.

A series of t-tests conducted by comparing allergy predictions on the final trial confirmed the observation that the oldest group were particularly compromised in terms of the NPP. The Young group's responses to compounds were significantly different to responses to elements in both the PPP ($t = 7.09$, $df=19$, $p<0.001$) and NPP ($t = 2.87$, $df=19$, $p<0.02$), as were the YO group's (PPP: $t = 4.75$, $df=17$, $p<0.001$; NPP: $t = 4.75$, $df=17$, $p<0.001$) whereas the OO group's responses in the PPP were significantly different ($t =5.61$, $df = 7$, $p<0.002$), but not the NPP ($t = 0$, $df=7$, $p>0.05$). This again demonstrates that Age decrements in the present experiment were confined to the OO group's responses to the NPP and that their predictions for this problem were no better than chance.

6.5.5: Rule Induction

As in Experiment 2 data were also analysed in terms of whether participants had correctly induced a rule of conjunction to help them solve the problem. In terms of age those who induced the rule correctly (RC) were younger (Mean Age 47.33, S.D. 20.96) than those who did not (RI: Mean Age 52.32, S.D. 24.34), but this difference was not statistically significant ($t= .650$, $df =44$, $p>0.05$). The standard deviations also indicate that both groups' ages were far from homogenous. Frequency counts indicate that 6 of 12 participants in the YO group and 1 of 8 in the OO group correctly induced the rule. For the younger group 6 participants were correct, and 14 incorrect. This indicates that the extra trials and design simplification helped older groups to give more accurate responses and consequently to induce the rule of conjunction. In this experiment the RC group scored significantly better on AH4 Total ($t= 3.89$, $df =35$, $p<0.001$) and DC ($t= 2.05$, $df =35$, $p<0.05$). Here the differences between groups were more significant for AH4 scores, suggesting that this factor was more important in determining rule induction than processing speed.

Analysis was again by two ANOVAs; one was Rule (2: RC & RI) by Problem (2) by Blocks (5) in terms of discrimination scores and the other Rule (2) by Stimulus (2) by Blocks (5) for accuracy scores. Both analyses yielded a significant effect of Rule ($F_{(1,44)}$=15.45, p<0.001) and of Blocks ($F_{(4,176)}$=41.32, p<0.001) and a Blocks by Rule interaction ($F_{(4,176)}$=5.82, p<0.001: see Figure 6.11). This demonstrates that the RI group learned the problems more quickly and accurately despite the lack of consistent age differences.

Figure 6.11: Rule by Blocks

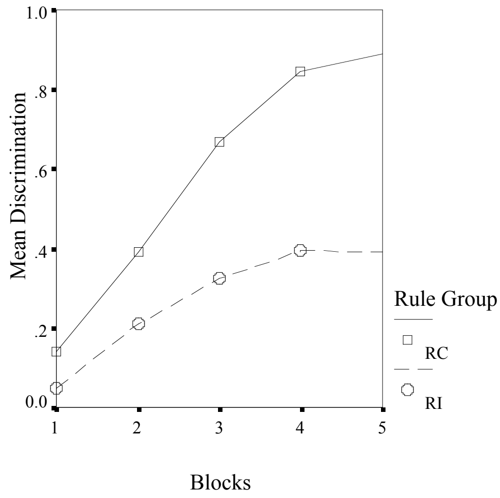
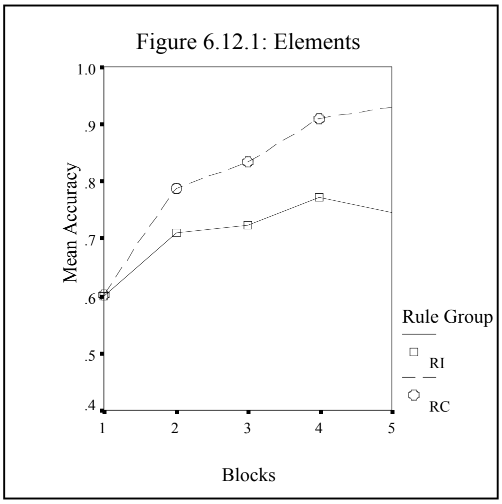


Table 6.17: Summary Statistics for Figure 6.11

	Rule Correct		Rule Incorrect	
Block	Mean	Std Deviation	Mean	Std Deviation
1	.14	.31	.05	.22
2	.39	.26	.21	.32
3	.67	.23	.32	.33
4	.85	.26	.39	.37
5	.89	.29	.39	.37

The Problem analysis demonstrated no significant effects or interactions beyond those alluded to above. The Stimulus analysis, however, revealed a main effect of Stimulus ($F_{(1,44)}$=13.12, p<0.002), and interactions between Rule and Stimulus ($F_{(1,44)}$=4.46, p<0.05) and Stimulus and Blocks ($F_{(4,176)}$=2.9, p<0.03: see Figures 6.12.1 and 6.12.2, overleaf). This suggests that although overall elements were learned better and quicker than compounds this difference was bigger for the RI than the RC group.



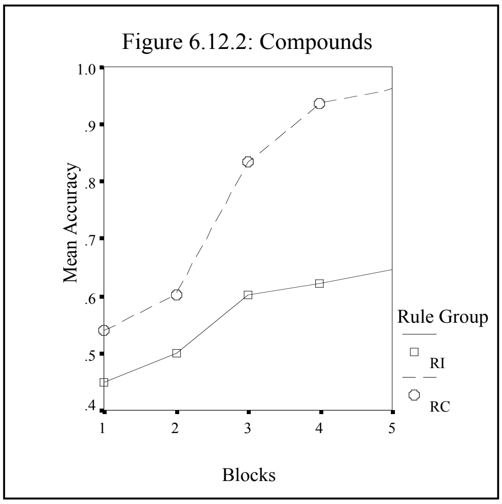


Table 6.18: Summary Statistics for Figures 6.12.1 & 6.12.2

Rule Group	Stimulus	Young		Young Old		Old Old	
		Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Rule Correct	Elements	.83	.05	.79	.17	.85	*n* = 1
	Compounds	.76	.11	.79	.13	.73	*n* = 1
Rule Incorrect	Elements	.72	.16	.71	.16	.69	.13
	Compounds	.64	.18	.52	.13	.48	.12

6.5.6: Multiple Regression

Once more an initial correlation was performed between potential predictor variables (Age, Years of Education (YE), Digit Cancellation (DC), MacQuarrie Total (McQ), MHV, and AH4 Total) and dependent variables (overall (OA) and final trial (FTA) accuracy). DC (r=0.367, p<0.03) and AH4 Total (r=0.401, p<0.02) correlated significantly with FTA. In terms of OA there were significant correlations with Age ($r=-0.332$, $p<0.03$), DC ($r=0.404$, $p<0.02$), McQ ($r=0.484$, $p<0.02$), and AH4 Total ($r=0.496$, $p<0.003$). As before only these significant correlations will be entered into the multiple regression equations using the enter method.

Consequently two predictor variables were entered as predictors of FTA: DC and AH4 Total. The model proved a significant predictor of FTA ($F_{(2,34)}=4.58$, $p<0.02$, adjusted $R^2 = 0.166$) although neither DC nor AH4 Total emerged as a single significant predictor.

For OA Age, DC, McQ, and AH4 Total were entered as predictor variables. Again the model proved significant ($F_{(4,11)}=4.49$, $p<0.025$) and was able to predict more variation in the dependent variable than the previous model (adjusted $R^2 = 0.482$). In this analysis Age emerged as the sole significant predictor of OA (beta = -0.555, $p<0.05$). This implies a slower start for older participants since Age did not even correlate with, let alone predict FTA.

6.5.7: Discussion

In this experiment, the overall age differences observed in Experiment 2 disappeared but the differences in participants' responses to stimuli and problems were significant as well as the interaction between Age and Problem. These observations were similar to those seen in Experiment 1 in that the OO group had difficulties in terms of learning a NPP relative to the other groups and participants overall found elements easier to learn than compounds. Again, this fits the idea that the oldest participants overgeneralise between stimuli and that linear solubility is a factor in determining the 'complexity' of a problem and may be consistent with FL decline. On the other hand the fact that compounds were more poorly learned than elements suggests a potentially MTL mediated deficit.

AH4 scores did not render the Age by Problem interaction non-significant, suggesting that NPPs' relative difficulty in this experiment is more the result of poorer basic learning processes than a failure to engage higher level configural processes. Overall, relative to Experiments 1 and 2 the present experiment shows that as problems become easier and less complex and participants are given more time to learn them then any age related deficits become smaller and are confined to the over seventy fives. The question that remains is; why should there be no age interactions in Experiment 2 when there were in Experiments 1 and 3? Perhaps this is a case of difficulty in that Experiment 1 was very difficult for all participants and produced a range of age related interactions whereas the simpler Experiment 2 just produced an overall age difference. It may be that the further simplified Experiment 3 led to deficits only for the OO group in terms of the NPP rather than the monotonic differences seen in Experiment 2, and that age itself is a factor in this above and beyond the effects of fluid intelligence. Perhaps the inability of AH4 scores to attenuate the Age by Problem interaction has more to do with compromised basic learning ability in a relatively simple problem, and this explains why Age emerged as the sole individually significant predictor of accuracy over the experiment, suggesting that basic HCL ability may be dissociable from more general abilities.

In terms of rule learning the present experiment showed that, given a simple enough problem and more trials to learn it, older adults are capable of inducing rules to help them solve HCL problems although the over seventy fives were under represented in the RC group. In this instance, the group differences between RC and RI groups in terms of AH4 Total and DC persisted, although differences were more marked in AH4 Total scores. This suggests that general intelligence in particular, but also perceptual speed, may mediate rule induction in all age groups. Again, the RC group learned the problem more quickly and thoroughly than the RI group, supporting the contention that rule induction in HCL problems is contingent on initial learning. The Rule by Stimulus interaction further supports this suggestion, and demonstrates that only the RI group had problems with compound learning relative to element learning. It may be that the difference in generalisation processes implied by this observation underlies the learning ability of the RC group and their capacity to induce rules successfully.

6.6: Experiments 4 and 5

Experiments 4 and 5 are both direct replications of Experiment 3 in that they both feature two concurrent patterning problems with bivalent outcomes (i.e. Allergy/No Allergy). The manipulation here is in the fact that Experiment 4 features two NPPs whereas Experiment 5 includes two PPPs. This is in order to ascertain whether the presentation of two different problems concurrently constitutes complexity in a HCL problem, to see whether learning of PPPs alone is easier than NPPs alone, and to establish the extent of learning that one can expect of older participants in order to design subsequent and more rigorous multiple stage experiments (see Experiments 9 & 10). Only the two older age groups participated in these experiments because younger participants will inevitably learn them well, and the main aim here was to simply see if older people could be expected to learn concurrent problems of a similar nature.

6.6.1: Participants

Participants in Experiment 4 were 10 YO volunteers with a mean age of 66.52 (SD=3.63) and 6 OO people with a mean age of 77.26 (SD=3.58). In Experiment 5 the YO group consisted of 11 participants with a mean age of

69.39 (SD=4.19) and the OO group were 7 volunteers with a mean age of 79.06 (SD=3.58).

Table 6.19: Participant Summary Statistics Experiment 4

	Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation
Age	66.52	3.63	77.26	3.58
Years of Education	13.00	3.43	15.17	3.60
AH4 Total	72.44	20.29	83.83	22.30
Digit Cancellation	16.89	3.30	16.33	4.68
MHV	34.33	3.83	35.00	6.39
MacQuarrie Total	105.83	22.64	114.50	31.09

Table 6.20: Participant Summary Statistics Experiment 5

	Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation
Age	69.39	4.19	79.06	3.58
Years of Education	12.91	2.55	14.14	2.41
AH4 Total	58.70	16.29	59.00	10.04
Digit Cancellation	16.50	6.08	17.00	4.47
MHV	33.38	2.83	36.00	4.83
MacQuarrie Total	99.13	26.69	88.00	22.35

6.6.2: Design & Materials

As stated the design of Experiment 4 featured two concurrent NPPs and Experiment 5 two concurrent PPPs. The foods used are detailed in Table 6.21 and 6.22, below.

6.6.3: Procedures

Procedures for both Experiments 4 and 5 were as outlined for Experiment 3.

Table 6.21: Experiment 4 Design

Abstract	*Version 1*	*Version2*	*Outcome*
A	Aubergine	Cheese	Allergy
B	Lettuce	Salmon	Allergy
C	Chicken	Apple	Allergy
D	Peaches	Carrots	Allergy
AB	Aubergine Lettuce	Cheese Salmon	No Allergy
CD	Chicken Peaches	Apple Carrots	No Allergy

Table 6.22: Experiment 5 Design

Abstract	*Version 1*	*Version2*	*Outcome*
A	Aubergine	Cheese	No Allergy
B	Lettuce	Salmon	No Allergy
C	Chicken	Apple	No Allergy
D	Peaches	Carrots	No Allergy
AB	Aubergine Lettuce	Cheese Salmon	Allergy
CD	Chicken Peaches	Apple Carrots	Allergy

6.6.4: Results: Experiment 4

Initial analysis for both experiments was by Age (2) by Stimulus (2) by Blocks (5) ANOVA. In Experiment 4 there were no significant effects of Age or Stimulus but an overall effect of Blocks was significant ($F_{(4,56)}$=22.51, $p<0.001$). There was also a significant Blocks by Stimulus interaction ($F_{(4,56)}$=2.67, $p<0.05$). Figures 6.13.1 and 6.13.2 suggest that this is the result of quicker initial learning of elemental stimuli and a drop in compound accuracy toward the end.

Figure 6.13.1: Experiment 4 Elements

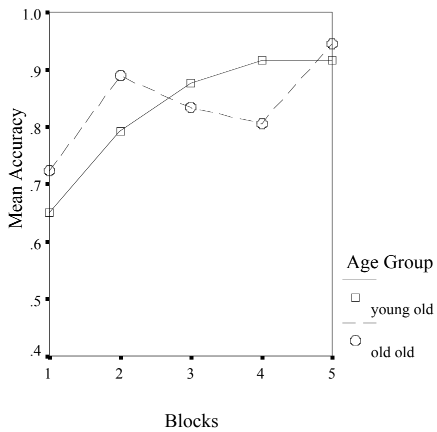


Figure 6.13.2: Experiment 4 Compounds

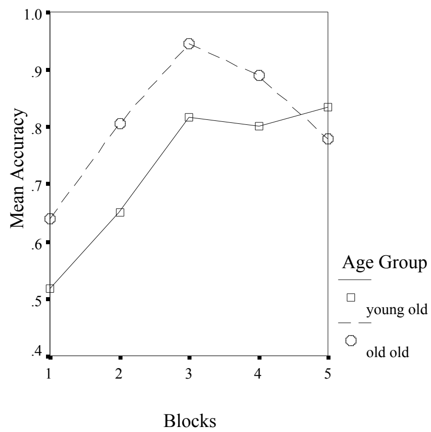


Table 6.23: Summary Statistics for Figures 6.13.1 & 6.13.2

	Elements (Fig. 6.13.1)				Compounds (Fig. 6.13.12)			
	Young Old		Old Old		Young Old		Old Old	
Block	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.65	.19	.52	.28	.64	.25	.72	.25
2	.79	.17	.65	.33	.81	.25	.89	.17
3	.87	.21	.82	.30	.94	.09	.83	.26
4	.92	.14	.80	.33	.89	.17	.81	.30
5	.92	.14	.83	.27	.78	.34	.94	.09

These data suggest that both age groups are capable of learning two concurrent negative patterning problems. To test this assumption and as in previous experiments t-tests were performed to find out if participants consistently discriminated between elements and compounds on the final trial of the experiment. The t-tests proved significant for both YO ($t=12.33$, $df=9$, $p<0.001$) and OO ($t=4.74$, $df=5$, $p<0.006$) groups.

Figure 6.15: Age by Blocks

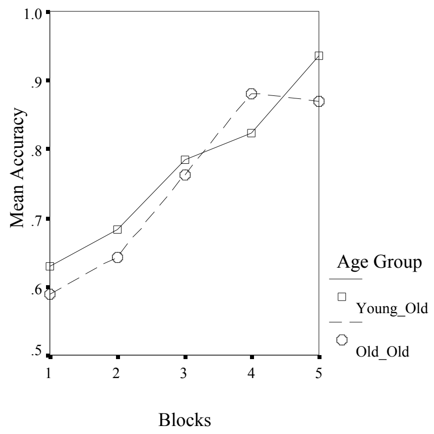


Figure 6.16: Experiment 5 Age by Stimulus

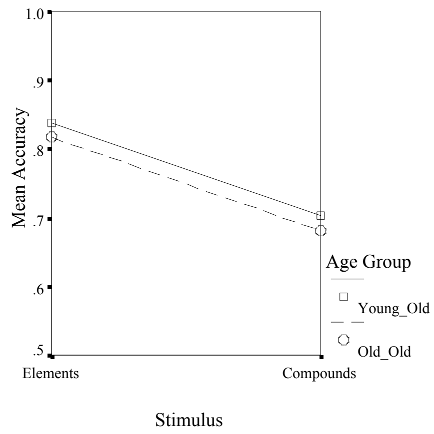


Table 6.24: Summary Statistics for Figure 6.15

Block	Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation
1	.26	.40	.18	.27
2	.36	.30	.29	.37
3	.57	.39	.52	.37
4	.64	.38	.76	.35
5	.87	.27	.74	.37

Table 6.25: Summary Statistics for Figure 6.16

Stimulus	Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation
Elements	.84	.12	.82	.13
Compounds	.70	.18	.68	.18

6.6.7: Results: Experiment 5

Initial analysis was, once more by Age (2) by Stimulus (2) by Blocks (5) ANOVA. There was the usual main effect of Blocks ($F_{(4,64)}$=15.87, p<0.001: see Figure 6.15) as well as a more interesting main effect of stimulus ($F_{(1,16)}$=13.48, p<0.003: see Figure 6.16) but no main effect of Age or any further interactions.

These results clearly show that learning two concurrent PPPs was relatively straightforward even for the over seventy fives. Since there were obviously no consistent Age effects a single t-test established that participants gave significantly different responses to elements and compounds on the final trial (t=8.04, df=17, p<0.001), indicating that the problems were well learned.

6.6.8: Discussion of Experiments 4 and 5

These experiments confirm that older participants are capable of learning a negative or positive patterning problem, providing only one problem type is presented at a time. This, in turn, means that presenting different problems concurrently constitutes complexity in HCL and presumably takes up more processing resources than presenting the same problem types concurrently. This suggests that configural generalisation processes take up limited resources that may be replaced by less resource intensive elemental processes as task complexity increases. There is also further support for the notion that elements are easier to learn than compounds in both Experiment 4 and 5, indicating that age related mnemonic decline might play a part. The absence of age effects in either experiment, however, indicates that age had little impact in learning similar concurrent HCL problems, even NPPs. As a consequence one can assume that it is not unreasonable to expect that older participants can learn two concurrent NPPs and PPPs, provided that problem types are similar.

6.8: Discussion of Positive and Negative Patterning Experiments

Overall, the results of Experiments 1 to 5 have largely been as expected. Firstly, and probably most importantly in the context of the present research, they have confirmed that a paper and pencil version of the food allergy task supports learning, and that older participants are capable of learning simple problems. Beyond this they have confirmed that in two of three experiments NPPs have been more difficult to learn than PPPs when presented concurrently, especially for older age groups. The NPP is elementally insoluble whereas the PPP may be soluble by adopting a threshold of activation rule, so this observation makes sense in terms of the notion that older people may overgeneralise between stimuli, although it is not clear exactly *why* they overgeneralise. Either overgeneralisation may occur in the Pavlovian (1927) or Hebbian (1949) sense because participants know the correct response but become confused between stimuli, or in the sense articulated by Lashley and Wade (1946) in that they can encode stimuli but become confused over the appropriate response. The MTL probably mediates the former, and MTL decline was suggested as a major reason for age related memory decline by Naveh-Benjamin (2000; Naveh-Benjamin et al. 2002; Naveh-Benjamin et al. 2003, Naveh-Benjamin et al. 2004). There is evidence from the present experiments that learning compound stimuli was more difficult for participants, especially older ones, which is consistent with this view, although not unequivocally since learning compounds may simply require more processing resources than learning elements, and this interpretation is reinforced by the observation that AH4 scores rendered differences between stimulus types non-significant when entered as a covariate. In addition there was a more general age related decline in learning ability, consistent with the latter conception of overgeneralisation mediated by the use of either elemental or configural strategies. Furthermore, there was evidence to suggest that greater ‘complexity’ in terms of number of outcomes and problems led to greater age-related deficits in learning. Moreover, when processing demands were further reduced by presenting concurrent similar problems (Experiments 4 and 5) age differences were heavily diminished relative to the learning of two similarly concurrent but different problems (Experiment 3). Since the memory demands of Experiments 3, 4, and 5 were identical one would not expect greater overgeneralisation in Experiment 3 as a result of MTL declines. Rather, they may be seen as indicative of age related declines in strategic, effortful, executive processing.

The multiple regression analyses were inconclusive but Age itself emerged as the most consistent predictor of accuracy in Experiments 2 and 3. Other than this, YE predicted accuracy for OA in Experiment 1. This may suggest that associative learning may be dissociable from other age related cognitive abilities, although DC consistently appeared in the significant models' predictive accuracy in Experiments 1 to 3, although not as individually significant factors. This may lend limited support for Salthouse's (1996) processing speed theory. There is also the observation that AH4 scores failed to attenuate age differences in learning completely when entered as a covariate, suggesting that very basic learning processes may be independent of higher order cognitive abilities. Beyond this there is little to tell concerning associative learning and ageing since the intention of these experiments was to confirm the suitability of the paradigm and describe any age related deficits in learning NPPs and PPPs, which they have achieved.

In terms of rule induction, these experiments have done nothing to disconfirm Shanks and Darby's (1998) suspicion that thorough learning of contingencies underlies rule learning. Age differences were observed between RC and RI groups in Experiment 2, where there were overall age related deficits in learning, but not in the simpler Experiment 3, where age related deficits were more specific to stimulus or problem type. Those in the RC groups learned the problems more quickly and thoroughly than RI groups in all experiments. Individual differences were also evident in terms of especially AH4 Total but also DC between RC and RI groups in Experiments 2 and 3. This suggests that rule induction may be mediated by more general, fluid abilities, and gives some support to theories suggesting that cognitive abilities are subsumed by factors that are more general. For Experiments 2 and 3 the full rule of conjunction stating that elements and compounds were always associated with opposing outcomes was necessary to be correct. This is clearly a demanding task that could tap into effortful processing, and rule learners' higher AH4 scores may reflect greater 'executive', frontally mediated abilities within this group. As a further observation, it may be that the relative ease with which participants learned the concurrent NPPs and PPPs in Experiments 4 and 5 may be due to the induction of a very simple rule that allowed participants to learn that all elements made the same predictions, and that all compound predictions should oppose them.

The experiments in this chapter have described age related differences in learning two particular types of non-linear problem. The next chapter extends the description of age related changes in associative learning to biconditional and conditional problems in order more fully to compare problems that have elemental solutions with those that do not.

Chapter 7: Formative Experiments II: Conditional and Biconditional Problems

7.1: Introduction

So far age related decline has been investigated only in terms of negative and positive patterning problems. While this has been a useful exercise there are some drawbacks to only using these problems. Firstly all problems contain twice as many elements as compounds, making meaningful comparisons between stimulus types difficult. It is certainly possible that the stimulus effects observed in experiments one to five were due to the high frequency of element trials rather than any particular difficulties with learning compound stimuli, although note that there were no more trials of any particular stimulus type than another so to benefit from this advantage participants would have had to establish stimulus-stimulus associations in order to generalise from one element to another. Another contamination involves the possibility of rule induction. Whilst this is an interesting phenomenon in its own right it could be considered a confounding variable when one is considering purely associative learning. The next three experiments are formative studies involving biconditional and conditional problems. Recall that a biconditional problem (e.g. AB+, CD+, AD-, BC-) is composed entirely of compounds consisting of two elements and that each element is associated both with an outcome and no outcome. The problem is therefore doubly difficult in that it only involves compound learning and is entirely non-linear. Furthermore there are no short cuts, heuristics or rules to help one master biconditional discriminations. Conversely a conditional problem (e.g. AC+, AD+, BC-, BD-), though also involving only compounds, is elementally soluble since only one element in each compound consistently predicts a response. This problem is, therefore, linearly soluble and should present far fewer problems for participants. Indeed this prediction has been confirmed in both animals (e.g. Saavedra, 1975) and humans (e.g. Shanks, Charles, Darby, and Azmi, 1998). This raises the question of whether older participants will be particularly disadvantaged when given a biconditional problem relative to a conditional problem.

7.2: Experiment 6

Experiment 6 featured a biconditional and a conditional problem presented concurrently. A conditional problem of the form AC+, AD+, BC-, BD- would be elementally soluble since elements A and B are consistent predictors of the outcome and would consequently acquire associative strengths of 1λ and 0λ, respectively.

Similarly a unique cue model would easily solve the problem by conceiving of it as an ACW+, ADX+, BCY-, BDZ- problem and allowing the unique cues W, X, Y, and Z to acquire associative strength. Note, however, that this is not necessary and the model can seem unparsimonious and inelegant under such conditions, since the problem is elementally soluble anyway and the decision of whether a unique cue is formed or not is arbitrary.

Pearce's model, however, makes the unequivocal prediction that the problem should be learned. In this instance compounds AC and AD would, since they generalise associative strength from each other, only need to reach 0.75λ and compounds BC and BD would reach asymptote at -0.25λ. This can be demonstrated by the calculations $V_{AC} / V_{AD} = 0.75 + (0.5 \cdot 0.75) + (0.5 \cdot -0.25)$ and $V_{BC} / V_{BD} = -0.25 + (0.5 \cdot -0.25) + (0.5 \cdot 0.75)$, which ensure that with appropriate generalisation AC and AD would have net associative values of 1λ and BC and BC net associative strengths of 0λ. Note that relatively little learning needs to occur for the problem to be learned, and as a consequence the Pearce (1987, 1994) model suggests that this problem would be solved quickly and easily.

The biconditional problem is another matter. A basic elemental model such as Rescorla-Wagner would simply predict that all elements would acquire an associative strength of 0.5λ since they predict an outcome or no outcome equally frequently, and therefore no discrimination between compound stimuli would occur.

A unique-cue approach would, on the other hand, allow a solution by assuming that the problem is represented as ABW+, CDX+, ACY-, and BDZ-. Here the unique cues W and X would acquire an associative strength of 1λ as the only consistent predictors of an outcome, and all other elements values of 0λ and the model would therefore successfully predict the correct outcomes for each compound stimulus. Note that within the unique cue framework the biconditional problem should be no more difficult to learn than a conditional problem.

Pearce's (1987, 1994, 2002) theory, on the face of it, would have great difficulty solving a biconditional problem. Each compound would generalise no associative strength from the other compound leading to a similar outcome but half of the associative strength of each of the compounds leading to an opposing outcome. If, for instance, one assumed that AB+ and CD+ had acquired associative strengths of 1λ then stimuli AC- and BD- would have to acquire associative values of -1λ in order to predict 0λ. This would mean that AB+ and CD+ would then have to acquire an associative strength of 2λ to counteract the generalisation from AC- and BD-, and so on, ad nauseam. As it stands, the model assumes judgements about similarity are based purely and simply on perceptual similarity, or the extents to which stimuli share superficial features. Given the well established empirical demonstrations regarding the influence of generalisation through perceptual similarity on discrimination learning (e.g. Guttman & Kalish, 1956; Hanson, 1959; Spence, 1937) this is not an unreasonable assumption, but there has to be more to generalisation than just perceptual similarity otherwise animals and humans would not be able to learn biconditional problems at all. Given the empirical demonstrations of animals' (e.g. Saavedra, 1975) and humans' (e.g. Shanks, Charles, Darby, & Azmi, 1998) ability to learn biconditional discriminations there is a clear need for modification of Pearce's (1987, 1994, 2002) theory, and a solution is possible by making one minor assumption about the nature of similarity.

When a problem is intractable because of perceptual overgeneralisation it is not unreasonable to suggest that solution may entail the engagement of higher cognitive processes in the form of inhibition of generalisation processes based on perceptual similarity. If, under these circumstances or if generalisation processes led to a stimulus losing all of its associative strength, one assumed that generalisation of associative strength through perceptual similarity was halved the biconditional problem becomes tractable. Here, the model would predict that compounds AB and CD would reach an associative strength of 1.33λ and compounds AC and BD –0.66λ since $V_{AB}/V_{CD} = 1.33 + (0.5 \cdot (0.5 \cdot -0.66)) + (0.5 \cdot (0.5 \cdot -0.66))$, and $V_{AC}/V_{BD} = -0.66 + (0.5 \cdot (0.5 \cdot 1.33)) + (0.5 \cdot (0.5 \cdot 1.33))$. Whilst this modification may seem a weakness of Pearce's model, it is still more elegant than the relatively unwieldy and unrealistically slow Gluck et al. (1993) model. Moreover, the assumption that fallacious judgements based on perceptual similarity can be attenuated by inhibition of generalisation is theoretically plausible and less arbitrary than the threshold assumptions that allow elemental solution of positive patterning problems, or the assumption that 'unique cues' attach themselves to compounds, thereby suspending all generalisation between perceptually similar stimuli. Furthermore, the assumption that learning a biconditional problem engages effortful inhibitory processing resources leads, quite naturally, to the prediction that older participants should find this problem particularly difficult relative to the conditional problem. Although this does not distinguish between elemental and configural models broadly, it is possible to discount unique cue solutions if, as predicted by the Rescorla-Wagner and Pearce theories, the conditional problem proves to be easier to learn than the biconditional problem.

It is anticipated, therefore, that young participants should easily learn the conditional problem and that the biconditional problem requires the engagement of inhibitory processes to prevent overgeneralisation between stimuli and should, as a consequence, be tractable but more slowly learned. For older participants the conditional problem should be soluble since it merely requires elemental strategies for solution. On the other hand the biconditional problem should be more challenging for these groups since they require engagement of executive processing resources or, possibly, the creation of unique cues for solution to be possible. Note that both problems make the same demands in terms of basic memory processes since they have the same number of compounds and outcomes to learn.

7.2.1: Participants

Participants were 30 undergraduates ('young' group) who completed the experiment as part of a course requirement. Their mean age was 23.03 (S.D. =

7.22) and ranged from 18.69 to 46.68 years. Older participants were 35 volunteers who were subdivided into a 'young old' (YO) group with a mean age of 68.19 (n = 25, S.D. = 4.36; range = 57-74.49) and an 'old old' group (OO) with a mean age of 77.81 (n=10, S.D. = 2.76, range = 75.04-83).

Table 7.1: Participant Summary Statistics

	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Age	23.03	7.22	68.19	4.36	77.81	2.76
Years Education	14.00	.00	10.64	1.63	12.60	3.03
AH4 Total	88.10	14.64	61.75	16.46	77.25	12.58
Digit Cancellation	21.93	4.33	14.88	3.44	14.50	4.12
MHV	27.03	3.59	30.52	3.78	34.30	3.43
MacQuarrie Total	.	.	98.00	30.18	96.40	26.26

7.2.2: Design & Materials

Table 7.2 shows the basic experimental design, which consisted of concurrent conditional and biconditional problems. Participants received fifteen blocks of 8 trials randomised within blocks, making 120 trials in total.

Table 7.2: Experimental Design

Biconditional				Conditional			
Abstract	Version 1	Version2	Outcome	Abstract	Version 1	Version2	Outcome
AB+	Milk Eggs	Fish Banana	Allergy	**EF+**	Fish Banana	Cheese Milk	Allergy
CD+	Chocolate Cheese	Avocado Olive Oil	Allergy	**EG+**	Fish Olive Oil	Cheese Chocolate	Allergy
AC-	Milk Chocolate	Fish Avocado	No Allergy	**HF-**	Avocado Banana	Eggs Milk	No Allergy
BD-	Eggs Cheese	Banana Olive Oil	No Allergy	**HG-**	Avocado Olive Oil	Eggs Chocolate	No Allergy

7.2.3: Procedures

Procedures were as described in the general procedure section of Chapter 6.

7.2.4: Results: Initial Analysis

Data were first analysed with an Age (3) by Problem (2) by Blocks (5) mixed ANOVA. This analysis showed no significant effects of Age or Problem but yet another main effect of Blocks ($F_{(4,248)}$=10.35, p<0.001: see Figure 7.1) was apparent, merely showing that participants' responses changed over the experiment.

Figure 7.1: Blocks by Age

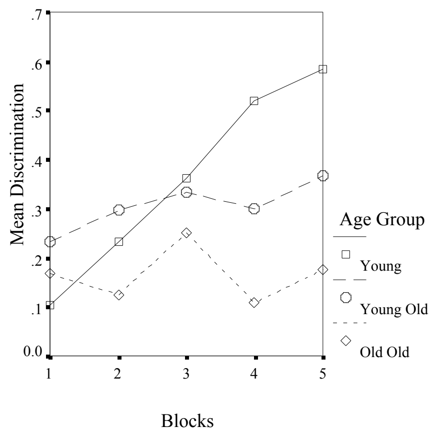


Figure 7.2: Problem by Blocks

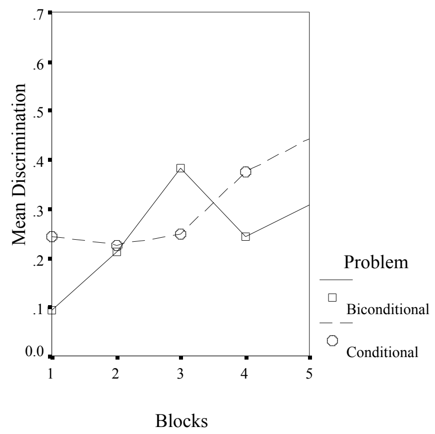


Table 7.3: Summary Statistics for Figure 7.1

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.10	.24	.23	.27	.17	.29
2	.23	.31	.30	.28	.12	.21
3	.36	.33	.33	.31	.25	.29
4	.52	.27	.30	.36	.11	.31
5	.58	.34	.37	.31	.18	.35

Table 7.4: Summary Statistics for Figure 7.2

Problem	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Biconditional	.33	.24	.25	.31	.17	.24
Conditional	.39	.36	.37	.31	.16	.26

There were, more interestingly, significant interactions between Age and Blocks ($F_{(8,248)}$=7.4, p<0.001) and between Problem and Blocks ($F_{(4,248)}$=6.74,

$p<0.001$) but other interactions proved non significant. The former results shows that older participants' responses changed less as the experiment progressed, Figure 7.1 confirms this interpretation, and this interaction remained significant once AH4 had been entered as a covariate ($F_{(8,220)} = 3.03$, $p<0.0035$). As for the latter interaction interpretation is more complex. As Figure 7.2 shows, discriminations for the conditional problem rose fairly systematically, whereas accuracy for the biconditional problem seemed more variable. This suggests that, on average, participants may have found the latter problem more confusing than the former, and a t-test showed that conditional discriminations were significantly greater than biconditional discriminations on the final trial ($t = 1.95$, $df=64$, $p<0.03$), although there had been no overall differences between the problems.

After this final trial responses were analysed with t-tests to see whether participants in each age group had significantly discriminated between stimuli associated with an outcome and those associated with no outcome (see Figure 7.3). This analysis showed that the Young group successfully learned both conditional ($t=7.21$, $df=29$, $p<0.001$) and biconditional ($t=6.16$, $df=29$, $p<0.001$) problems. The YO group learned the conditional ($t=5.48$, $df=24$, $p<0.001$), but not the biconditional problem ($t=1.25$, $df=24$, $p>0.05$). The OO group, on the other hand, learned neither the biconditional ($t=1.86$, $df=9$, $p>0.05$) nor the conditional problem ($t=0.67$, $df=9$, $p>0.05$).

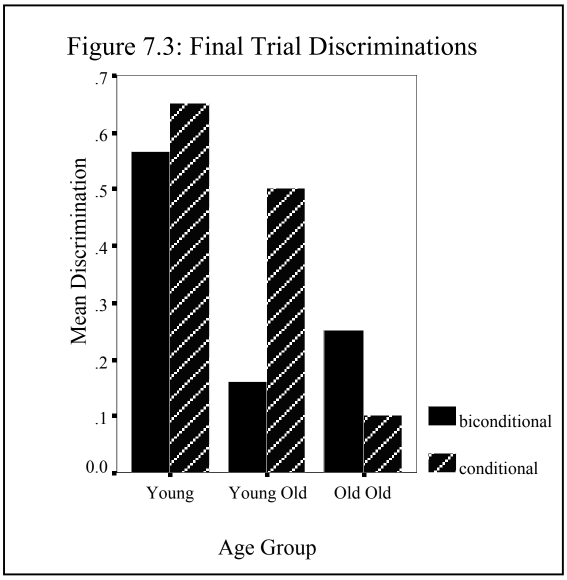


Table 7.5: Summary Statistics for Figure 7.3

	Young		Young Old		Old Old	
Problem	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Biconditional	.57	.50	.16	.64	.25	.42
Conditional	.65	.49	.50	.46	.10	.46

These data suggest that older participants may be slower to learn the problems than younger people and that ultimately the biconditional problem was less well learned than the conditional problem. The differences between the problems seems to have become more pronounced as the experiment progressed, implying that initial learning may have been difficult but that accuracy for the easier conditional problem improved progressively but the biconditional problem seems to have been confusing. This may have been due to attempts to learn both problems elementally.

7.2.5: Multiple Regression

In experiment 6 only Age ($r = -0.38$, $p<0.03$) and Years of Education (YE; $r = 0.27$, $p<0.03$) correlated significantly with FTA whereas none of the potential predictor variables were associated with OA. A single multiple regression analysis was performed using the enter method, therefore, on FTA with Age and YE entered as predictor variables. This model proved statistically significant overall ($F_{(2,62)}$=5.24, p<0.009; R^2 = 0.15), and Age emerged as the sole significant predictor of FTA (Beta = -0.34, p<0.03).

7.2.6: Discussion

The results of this experiment are largely as expected. Older participants learned more slowly and the biconditional problem was less well learned by all participants than the conditional problem. Although there were no Age by Problem interactions, by the final trial the YO group only discriminated successfully in the conditional problem, again suggesting that overgeneralisation between similar stimuli and tendency to use elemental assumptions may underlie their inability to learn the biconditional problem. The OO group, on the other hand, did not significantly discriminate between stimuli associated with an outcome and stimuli associated with no outcome in either problem, suggesting that this group's processing resources may have been overwhelmed by the concurrent problems leaving them unable to discriminate at all. The interpretation of a steady, monotonic age related decline in learning ability in the current experiment was supported by the finding that Age emerged as the only significant predictor of FTA, although no individual difference variables even correlated with OA. Taken together the results of the multiple regression analyses again suggest that age related decline in associative learning ability might be dissociable from other factors rather than subsumed by them. It is likely that the younger participants' ability to use a modified configural strategy involving partial inhibition of generalisation based on perceptual similarity underlies their superiority here. Although it is equally true that unique cue assumptions make solution of a biconditional problem possible in the current experiment this leads to the assumption that there should be no difference in terms of difficulty between biconditional and conditional problems. Given the Problem by Blocks interaction, therefore, it is unlikely that younger participants are pursuing any kind of elemental strategy, whereas the YO seem to be employing elemental strategies since they learned the configural but not biconditional problems, and the demands of learning both problems concurrently seemed to overwhelm the processing resources of the OO group since they failed to learn either problem. The observation that AH4 scores failed to attenuate the Age by Blocks interaction is problematic, but may reflect a slower basic learning rather than any decrement in higher order processing.

7.3: Experiments 7 & 8

One question Experiment 6 raises is whether older participants are capable of learning conditional and biconditional problems alone. This is important for the critical experiments of the next chapter since it gives an idea of what older participants can reasonably be expected to learn, and can guide design of multiple stage experiments that will be able to discriminate more clearly between the predictions of elemental and configural theories. Experiments 7 and 8, therefore, present YO and OO groups with single conditional and biconditional problems. As with Experiments 4 and 5 the methods and procedures for these experiments will be presented jointly, followed by separate analyses and discussions. Since the aims of these experiments are limited no multiple regression analyses will be performed.

7.3.1: Participants

Experiment 7 (biconditional problem) enlisted the help of 18 YO participants (Mean Age = 67.18, SD = 4.3) and 7 OO volunteers (Mean Age = 77.09, SD = 3.38). Participants in Experiment 8 (conditional problem) were 11 YO (Mean Age = 68.59, SD = 3.23) and 6 YO (Mean Age = 77.14, SD = 2.57) volunteers.

Table 7.6: Participant Summary Statistics Experiment 7

	Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation
Age	67.18	4.30	77.09	3.38
Years Education	12.47	2.81	13.00	3.87
AH4 Total	71.21	20.02	74.83	11.37
Digit Cancellation	16.93	3.99	15.83	4.54
MHV	32.89	4.43	34.00	5.97
MacQuarrie Total	105.39	22.97	101.86	38.38

Table 7.7: Participant Summary Statistics Experiment 8

	Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation
Age	68.59	3.23	77.14	2.57
Years of Education	12.60	2.32	13.83	4.12
AH4 Total	79.25	22.85	69.33	17.88
Digit Cancellation	16.13	3.31	16.67	4.32
MHV	32.27	5.53	34.17	2.48
MacQuarrie Total	117.36	30.61	97.00	28.93

7.3.2: Design

Experimental designs are shown in Tables 7.8 and 7.9 below. Note that different stimuli and responses have been used in these experiments but that the paradigm remains the same. In these experiments stimulus words were the signs of the zodiac.

Table 7.8: Experiment 7 Design

Abstract	Version 1	Version2	Outcome
AB +	Capricorn Aries	Scorpio Libra	Open
BC -	Aries Leo	Libra Taurus	Closed
CD +	Leo Pisces	Taurus Aquarius	Open
AD -	Capricorn Pisces	Scorpio Aquarius	Closed

Table 7.9: Experiment 8 Design

Abstract	Version 1	Version2	Outcome
AB +	Capricorn Aries	Scorpio Libra	Open
AC +	Capricorn Leo	Scorpio Taurus	Open
DB-	Pisces Aries	Aquarius Libra	Closed
DC -	Pisces Leo	Aquarius Taurus	Closed

7.3.3: Procedures

Participants were told that there were 'magic words' that either opened a door or left it closed via the following printed instructions that were verbally reinforced by the experimenter. Other than this all procedures were identical to those used in previous experiments: stimulus words were presented and read out, participants made their responses and then corrective feedback was given.

The learning task requires you to make a prediction about which magic words open a door, and which do not. You will be guessing at first but will become more accurate as the task progresses.

This is a hypothetical, artificial *categorisation task. Simply tick the box underneath the prediction (i.e. OPEN or CLOSED) you make for the magic words as they are presented on the screen. Each trial is numbered and you* *should match the numbers on your answer sheets with the numbers on the screen.*

When you have recorded each prediction look up so I know you have finished, otherwise the whole process will take much longer than necessary. Please don't make notes or crib sheets, the task is designed to be completed unaided and to do otherwise would invalidate the results.

7.3.4: Experiment 7 Results

Figure 7.4: Age by Blocks

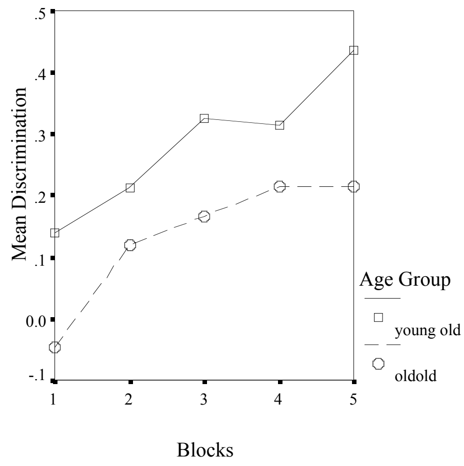


Figure 7.5: Final Trial Discriminations

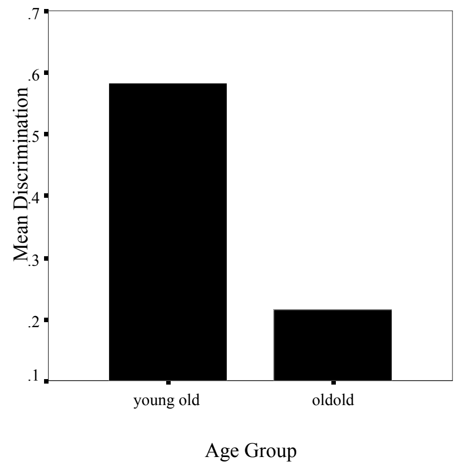


Table 7.10: Summary Statistics for Figure 7.4

Block	Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation
1	.21	.54	-.05	.27
2	.21	.32	.12	.27
3	.32	.37	.17	.33
4	.31	.48	.21	.27
5	.44	.40	.21	.30

Table 7.11: Summary Statistics for Figure 7.5

Young Old		Old Old	
Mean	Std Deviation	Mean	Std Deviation
.58	.46	.21	.27

Initial analysis was by an Age (2) by Blocks (5) ANOVA. This revealed only a significant main effect of blocks ($F_{(4,92)}=3.12$, $p<0.02$), indicating merely that participants' responses had changed as the experiment progressed. Figure 7.4 shows that this was due to an increase in accuracy, and suggests some age differences, although these were not significant.

As previously t-tests were then performed to see if participants in either age group had predicted significantly different outcomes for trials associated with outcomes and no outcomes. This showed that the YO had successfully discriminated between trial types on the final presentation ($t = 5.36$, $df = 17$, $p<0.001$), whereas the OO did not ($t = 2.12$, $df = 6$, $p>0.05$). This shows that although there were no significant age differences overall, by the final trial the YO group had learned the biconditional problem better than the OO group.

7.3.5: Experiment 8 Results

As in the previous experiment the conditional problem was first analysed using an Age (2) by Blocks (5) ANOVA. This yielded another significant effect of Blocks ($F_{(4,60)}=3.12$, $p<0.02$), although no other main effects or interactions were significant (see Figure 7.6).

Figure 7.6: Age by Blocks

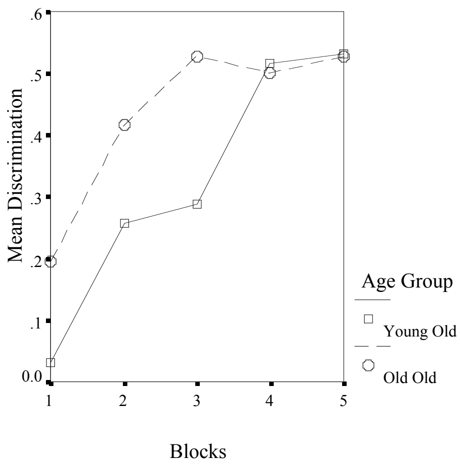


Table 7.12: Summary Statistics for Figure 7.6

Block	Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation
1	.03	.36	.19	.43
2	.26	.51	.42	.48
3	.29	.55	.53	.45
4	.52	.42	.50	.52
5	.53	.49	.53	.45

Another set of t-tests were performed separately for each age group to determine whether they had learned the conditional problem by the final trial. In this instance both YO ($t = 4.18$, $df = 10$, $p<0.003$) and OO ($t = 3.796$, $df = 5$, $p<0.015$) groups consistently discriminated between trials associated with outcomes and those associated with no outcome.

7.3.6: Experiment 7 & 8 Discussion

Overall these experiments have demonstrated that older participants are able to learn a single conditional problem when presented alone. On the other hand the oldest participants still had difficulty learning the biconditional problem. This reinforces the observations concerning age related deficits being particularly pronounced in non-linear problems and the relative difficulty of the biconditional problem. It also gives more credence to the notion that older people's associative learning deficits become more marked over the age of seventy-five, and may be due to overgeneralisation between stimuli and the inability to apply configural strategies. More importantly, these experiments show that one shouldn't assume that older participants are able to learn a biconditional problem. This will inform the design of Experiment 11 in the next chapter.

7.4: Discussion of Biconditional and Conditional Problems

One can assume that these experiments reflect participants' associative learning ability for linear and non-linear problems because of the lack of rules that could potentially be induced to aid learning. Experiment 6 showed that older participants could not learn a biconditional problem when presented concurrently with a conditional problem and Experiment 7 showed that even when a biconditional problem was presented alone the over seventy fives could not learn it after fifteen trials. This underlines the difficulty of the problem and provides further evidence that the ageing process results in participants overgeneralising between stimuli and attempting to use elemental strategies even when inappropriate. Younger participants are unlikely to have been using any kind of elemental strategies since they learned the biconditional problem, but more poorly than the conditional problem. This is in accord with configural rather than unique cue theories. Age predicted FTA in Experiment 6. This might be similar to the observations made in the previous chapter in that accuracy in the more difficult experiments tended to be predicted by age, and concurrent conditional and biconditional problems can be regarded, both theoretically and empirically, as difficult. Certainly it suggests, again, that associative learning accuracy may be dissociable from other cognitive abilities, particularly since AH4 scores failed to negate the Age by Blocks interaction in Experiment 6 when entered as a covariate, or that the background test batteries do not reflect the vital predictive factor.

The last two chapters have been largely descriptive. This has been a necessary step because of the lack of extant data relating to ageing and HCL tasks. The next chapter describes three critical experiments which look in more detail at the way ageing affects the learning process: whether ageing leads to greater overgeneralisation and the use of elemental strategies, if stimulus-stimulus learning is affected, and how rule induction shapes people's responses.

Chapter 8: Critical Experiments

The previous two chapters have dealt with an examination of what one can reasonably expect older adults to learn in an experimental situation using a human conditional learning task, and have made some contribution in terms of understanding the nature of any decline in generalisation processes with age. The following three experiments look at age differences in vulnerability to proactive and retroactive interference in learning using positive and negative patterning, and conditional and biconditional problems. In this sense elemental and configural associative learning theories make different predictions concerning the influence of prior learning on new learning (pro-active interference) and the effect of new learning on old learning (retroactive interference).

8.1: Experiment 9 Introduction

Experiment 9 consisted of three stages and was designed to test the extent of pro- and retro-active interference on associative learning as well as to look at the effects of rule induction. Stage 1 consisted of two concurrent PPPs (A-, B-, AB+; C-, D-, CD+), Stage 2 was composed of two concurrent NPPs, one of which featured novel stimuli (E+, F+, EF-) whereas the other was a revaluation of one of the Stage 1 problem stimuli (A+, B+, AB-), and the third stage reintroduced unreinforced Stage 1 stimuli in order to evaluate the effects of Stage 2 revaluation.

From earlier results (see Experiment 5) it should be the case that all age groups should be capable of learning the Stage 1 PPP contingencies, perhaps because the solution of two similar problems allows enough cognitive resources to remain for older participants to engage configural, or unique-cue processing rather than having to rely on elemental processes. Stage 2 contingencies, however, should be much more difficult for older participants since they involve reversal learning for one of the problems, which may, according to FL theory, lead to perseverative responses and pro-active interference. On the other hand Stage 2 contingencies may be easier for younger participants who may be able to transfer knowledge from how to solve Stage 1 to Stage 2, particularly since the PPP is easier than the NPP and pre-training on an easier discrimination facilitates learning a harder discrimination in rats (Mackintosh et al. 1970) and humans (Suret et al. 2003). Furthermore the younger participants' more flexible learning may enable them to similarly reverse a heuristic rule in a comparable way (c.f. Weisberg et al. 1981; Novick et al. 1991).

According to associative learning theories in the Test Stage younger participants should preserve Stage 1 C-, D-, CD+ responses whilst giving responses consistent with Stage 2 for A, B and AB stimuli. Older participants should be more prone to pro- and retro-active interference and should not discriminate as well or as consistently between problems. On the other hand it may be that rule learning, although contingent on associative learning, is general and participants may apply a rule as an heuristic to solve a problem, possibly resulting in revaluation of Stage 1 C, D, and CD contingencies to coincide with A, B, AB contingencies since participants may apply the last rule they learned to their unreinforced Test Stage predictions.

More formally in Experiment 9 the Rescorla-Wagner (1972) model predicts that in Stage 1 compound stimuli will acquire an associative strength of 1λ and elements an associative strength of 0.5λ. In Stage 2 the predictions would not alter since each element would still each have a value of 0.5λ since they are associated with an outcome as often as they are not. As a consequence there should be no difference between responses in Stage 1 and Test if participants are employing a simple elementally based rule of summation. Note that, even if participants manage to engage more cognitively demanding unique cue or configural processes in order to solve Stages 1 and 2, use of an elemental summation strategy in the test stage would result in very little or no discrimination at test.

A unique-cue model, on the other hand, would allow Stage 1 discriminations to be solved by assigning unique cues W and X to the compounds ABW+ and CDX+, which would be the only predictors of an outcome and, as a consequence, acquire an associative value of 1λ. Similarly, in Stage 2 new unique cues would be assigned to compounds ABY- and EFZ-, which would acquire associative strengths of -2λ to balance the elements' associative values of 1λ. Predictions are less clear in the test stage, and depend on whether one assumes that activation of stimulus CDX would lead to activation of ABW because compound CD forms part of contextual cue W, or if compound ABY would be activated by presentation of stimulus AB since this is the most recent example of AB. Effectively this suggests that participants' responses should either be consistent with Stage 1, in which case AB would have a value of 1λ, or it is equally likely that participants would respond to the AB compound as they did in Stage 2, in which case AB would attract responses consistent with an associative strength of -2λ. Clearly, though, C, D, and CD stimuli would be unchanged from Stage 1 and elements A and B should still have an associative value of 1λ. In many circumstances the unique cue model's ability to treat compounds entirely separately from their elements is helpful, but in this instance this property makes the predictions of the model less than clear.

As far as the Pearce (1987, 1994) model is concerned predictions for the first two stages are the same as detailed earlier for the PPP and NPPs in Experiment 1. Stage 1 PPPs would lead to A, B, C, and D elements acquiring an associative strength of -1λ while the compounds AB and CD would have a value of 2λ. In Stage two the NPPs would mean that elements A, B, E, and F would acquire a value of 2λ and the compounds AB and EF an associative strength of -2λ. This means that at test elements A and B will have a strength of 2λ, C and D a value of -1λ, AB an associative valence of -2λ, and CD a value of 2λ. As a consequence responses to C, D, and CD should be unaffected by Stage 2 discriminations whereas A, B, and AB responses should be consistent with Stage 2.

The question of the nature of age related decline in stimulus-stimulus associations will be tested in this and two further experiments with a compound recognition task. Participants will be asked whether they had seen a number of compounds or not during the course of the experiment. Stimulus compounds will include all compounds presented during the experiments together with the same number of novel compounds composed of the same elements, and the task will derive two scores: Hits and False Recognition. Hits denote the number of correctly identified compounds whilst False Recognition is the difference between the number of compounds incorrectly having been identified as seen and the number of hits. It should be expected that there will be a monotonic decline in performance with age. Note that, according to LaVoie et al. (2006), an index of False Recognition may be indicative of executive dysfunction and FL mediated overgeneralisation, whereas number of correct hits may give an indication of more purely mnemonic, MTL mediated ability.

8.1.1: Experiment 9 Participants

Participants for Experiment 9 were 44 Young undergraduates (Mean Age = 22.88, SD = 6.73), 23 YO volunteers (Mean Age = 67.04, SD = 4.18), and 9 OO contributors (Mean Age = 77.89, SD = 2.74).

Table 8.1: Participant Summary Statistics

	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Age	22.88	6.73	67.04	4.18	77.89	2.74
Years Education	14.00	.00	12.39	2.43	14.78	4.79
AH4 Total	79.68	19.61	82.94	16.18	62.63	14.7
Digit Cancellation	24.27	5.60	17.92	4.50	13.64	5.23
MHV	26.39	3.25	36.75	3.02	36.40	4.93
MacQuarrie Total	.	.	120.33	21.91	107.20	26.29

8.1.2: Design

Table 8.2: Experiment 9 Design

Stage 1			***Stage 2***			***Test***		
	1	2		1	2		1	2
A-	Chocolate	Olive Oil	A+	Chocolate	Olive Oil	A	Chocolate	Olive Oil
B-	Fish	Bread	B+	Fish	Bread	B	Fish	Bread
AB+	Chocolate Fish	Olive Oil Bread	AB-	Chocolate Fish	Olive Oil Bread	AB	Chocolate Fish	Olive Oil Bread
C-	Olive Oil	Cheese	E+	Cheese	Chocolate	C	Olive Oil	Cheese
D-	Bread	Avocado	F+	Avocado	Fish	D	Bread	Avocado
CD+	Olive Oil Bread	Cheese Avocado	EF-	Cheese Avocado	Chocolate Fish	CD	Olive Oil Bread	Cheese Avocado

Experiment 9 consisted of three stages: two with corrective feedback and a third test stage. Stage 1 consisted of two concurrent Positive Patterning Problems (PPP; A-, B-, AB+; C-, D-, CD+) presented in ten blocks of six trials. Stage 2 presented one of Stage 1's problems revalued as a Negative Patterning Problem (NPP; A+, B+, AB-) and a novel NPP (E+, F+, EF-), again presented in ten blocks of six trials. Following this, participants were given five blocks of six unreinforced test trials for stimuli A, B, C, D, AB, and CD. Table 8.2 (above) shows the design and stimuli used in the two versions of the experiment.

8.1.3: Procedures

Procedures were as detailed in Chapter 6. Each stage was presented in its entirety before a new stage was begun. Participants were not notified that the experiment would feature different stages nor were they told when Stage 1 ended and Stage 2 began. They were, however, informed that they would receive some unreinforced trials at the end of the experiment. This was explained as a way of testing how well they remembered what they had learned over the course of the experiment. Once the experiment was over participants were given a compound recognition task. This consisted of three compounds they had seen in the experiment (AB, CD, EF) and three they had not (AC, BE, CF) and participants simply had to indicate whether they had seen the compounds during the experiment. This derived two scores: 'Hits' (how many compounds correctly identified as having been seen or not seen) and 'False Alarms' (how many non-experimental compounds had incorrectly been identified as 'seen').

8.1.4: Experiment 9 Results

8.1.4.1: Stage 1 Analysis

Stage 1 results were analysed initially with an Age (3) by Blocks (5) ANOVA. This analysis revealed main effects of both Age ($F_{(2,73)} = 4.09$, $p<0.025$), and of Blocks ($F_{(4,292)} = 90.21$, $p<0.001$), but no interaction of Age and Blocks (see Figure 8.1). This simply shows that younger participants learned the problem almost perfectly, and older participants learned them significantly less well overall, although overall Bonferroni post hoc age group comparisons proved non-significant, and AH4 scores attenuated the Age effect when entered as a covariate ($F_{(2,68)} = 2.88$, $p>0.05$).

Final trial learning was tested in the usual way by performing t-tests to check that participants were discriminating significantly between elements and compounds. The tests showed that all groups had learned the problems by the end of Stage 1 (Young: t = 78.1, df = 43, p<0.001; YO: t = 9.65, df = 22, p<0.001; OO: t = 5.29, df = 8, p<0.002).

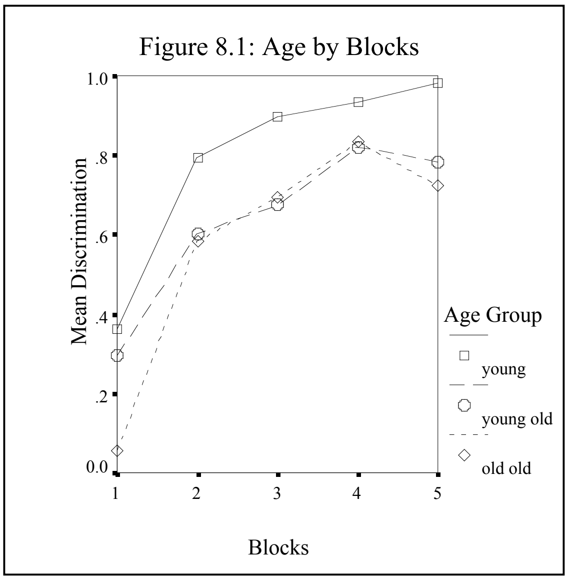


Table 8.3: Summary Statistics for Figure 8.1

	Young		Young Old		Old Old	
Block	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.36	.32	.29	.37	.06	.07
2	.79	.23	.60	.58	.58	.33
3	.90	.27	.67	.48	.69	.41
4	.93	.18	.82	.26	.83	.33
5	.98	.09	.78	.33	.72	.55

8.1.4.2: Stage 1 Multiple Regression

Of those individual difference variables identified in Chapter 5 Age and Years of Education (YE) correlated with Overall Accuracy (OA) (Age: r = -0.309, p<0.008; YE: 0.369, p<0.002) and the same variables correlated with

Final Trial Accuracy (FTA) (Age: r = -0.242, $p<0.04$; YE: 0.408, $p<0.001$). Age and YE were therefore entered into two multiple regression analyses using the enter method as predictor variables with OA and FTA as dependent variables.

For OA the model proved significant ($F_{(2,73)} = 9.11$, $p<0.001$, Adjusted $R^2 = 0.178$) with both Age (Beta = -0.26, $p<0.02$) and YE (Beta = 0.33, $p<0.004$) proving significant individual predictors of OA.

For FTA the model was, again, significant ($F_{(2,73)} = 9.04$, $p<0.001$, Adjusted $R^2 = 0.177$) however only YE emerged as an individually significant predictor of FTA (Beta = 0.379, $p<0.002$).

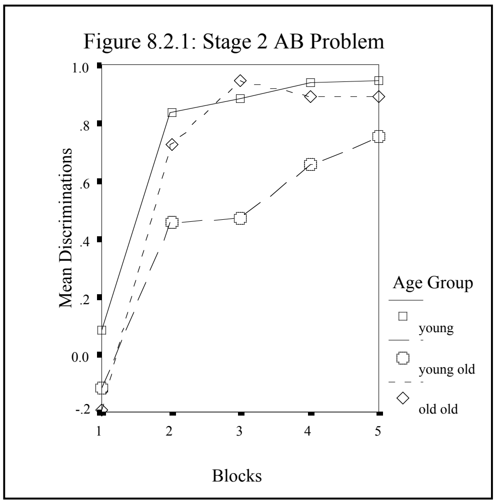
8.1.5: Results: Stage 2

8.1.5.1: Stage 2 Analysis

Stage two discriminations were analysed using an Age (3) by Blocks (5) by Problem (2: A+, B+, AB- (AB problem); E+, F+, EF- (EF problem)) ANOVA. This showed a significant main effect of Age ($F_{(2,73)} = 5.26$, $p<0.008$) and Bonferroni post-hocs showed that this difference was confined to that between Young and YO groups (mean difference = 0.26, $p<0.006$). Note, however, that with the introduction of AH4 as a covariate that the effects of Age ($F_{(2,68)} = 2.16$, $p>0.05$) was rendered non-significant. Figures 8.2.1 and 8.2.2 show that whilst participants' discriminations for the revalued AB problem were initially worse than those for the novel EF problem this difference became less as the experiment progressed, suggesting some proactive interference effects. It also, surprisingly, shows that the YO group were the worst at learning Stage 2 discriminations overall, a conclusion verified by the Bonferroni comparisons alluded to earlier and implying that this group were most vulnerable to proactive interference, perhaps because they had learned the Stage 1 discriminations more completely than the OO group and were therefore vulnerable to making perseverative errors.

Table 8.4: Summary Statistics for Figure 8.2.1

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.09	.46	-.12	.51	-.19	.51
2	.84	.42	.46	.57	.72	.26
3	.88	.40	.47	.54	.94	.11
4	.94	.31	.65	.45	.89	.22
5	.94	.31	.75	.53	.89	.22



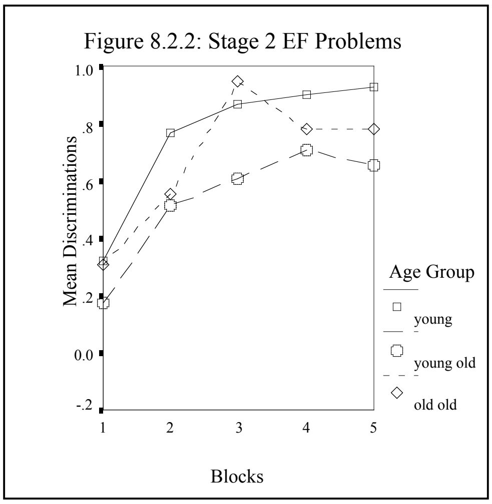


Table 8.5: Summary Statistics for Figure 8.2.2

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.32	.45	.17	.53	.31	.24
2	.77	.46	.51	.51	.56	.27
3	.86	.38	.61	.49	.94	.11
4	.90	.34	.71	.44	.78	.44
5	.93	.33	.65	.49	.78	.44

As previously t-tests were employed to see whether participants discriminated between elements and compounds on the final trial of Stage 2. In this instance the Young group's responses to stimuli were significantly different for both the AB problem ($t = 21$, $df = 43$, $p<0.001$) and the EF problem (t =

18.51, DF = 43, $P<0.001$). The YO group's responses were also significantly different (AB problem: $t = 6.28$, $df = 22$, $p<0.001$; EF problem: $t = 5.66$, $df = 22$, $p<0.001$), as were the OO Group's (AB problem: $t = 12.1$, $df = 8$, $p<0.001$; EF problem: $t = 5.29$, $df = 8$, $p<0.002$). This confirms that, although age differences were apparent, participants learned both discriminations by the end of the tenth trial.

8.1.5.2: Stage 2 Multiple Regression

For this analysis OA and FTA were calculated separately for the AB and EF problems because of the Blocks by Problem interaction identified earlier. For the AB problem OA correlated with Age ($r = -0.297$, $p<0.01$), AH4 Total ($r = 0.276$, $p<0.05$), and YE ($r = 0.237$, $p<0.04$) and FTA with YE ($r = 0.285$, $p<0.015$). For the EF problem OA correlated with Age ($r = -0.229$, $p<0.05$) and AH4 Total ($r = 0.311$, $p<0.025$), and FTA with Age ($r = -0.251$, $p<0.03$) and YE ($r = 0.24$, $p<0.04$). These predictor variables were entered into multiple regression analyses using the enter method.

In terms of the AB problem the predictors of OA were Age, AH4 Total, and YE. This model was significant despite only predicting a small amount of variance ($F_{(3,68)} = 2.98$, $p<0.05$, Adjusted $R^2 = 0.099$) and Age (Beta = -0.263, $p<0.05$) and AH4 Total (Beta = 0.268, $p<0.045$) were significant predictors of OA, although YE was not. For FTA YE was entered as a predictor and resulted in a significant model ($F_{(1,74)} = 6.56$, $p<0.015$, Adjusted $R^2 = 0.069$) and YE proved a significant individual predictor of FTA (Beta = 0.285, $p<0.015$). Presumably, this pattern of results suggests that overall accuracy reflects the effects of proactive interference, which may have been largely overcome by the final trial. This analysis is consistent with the Problem by Blocks interaction found in the previous section.

For the EF problem the pattern was slightly different. Here Age and AH4 Total were entered as predictors of OA and derived a near significant overall model ($F_{(2,69)} = 3.15$, $p = 0.051$, Adjusted $R^2 = 0.074$) with AH4 Total emerging as a significant individual predictor (Beta = 0.309, $p<0.025$). For FTA Age and YE were entered as predictor variables and this produced a significant model ($F_{(2,73)} = 4.23$, $p<0.02$, Adjusted $R^2 = 0.079$) but neither variable emerged as a significant sole predictor of FTA in the EF problem.

These results suggest that the YO group were most prone to proactive interference in this stage, presumably because they had learned the Stage 1 contingencies better than the OO group. Despite this all age groups ultimately learned the problems, although the AB problem was learned more slowly than the EF problem.

8.1.6: Test Stage Analysis

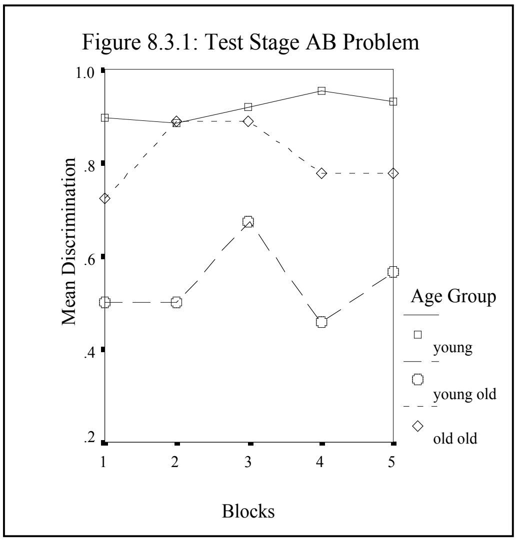
There are a number of questions to be answered through the test stage analysis. Firstly, do participants preserve the Stage 1 C-, D-, CD+ discriminations and differentiate between these and the revalued A+, B+, AB- problem learned in Stage 2 or does reverting to the context of Stage 1 lead them to revert to their earlier response patterns? Alternatively it may be that Stage 2 revaluation leads participants to employ the NPP solution as an heuristic, leading to C+, D+, CD- responses during the test stage. Secondly: are participants' responses consistent, indicating little confusion or uncertainty around responses? Thirdly, are test stage responses consistent with elemental, unique cue, or configural models of learning, and what can that tell us about the processes of generalisation being used? Fourthly, do older participants show a mnemonic decline in failing to correctly recognise compounds seen during the experiment, and, furthermore, do they falsely recognise unseen compounds as having been seen?

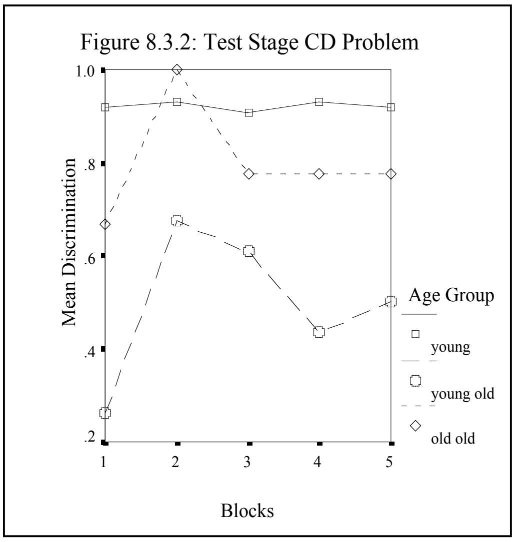
8.1.6.1: Test Stage Responses and Consistency

A visual inspection of the pattern of responses during data entry suggested that Young participants consistently responded to both A, B, AB and C, D, CD stimuli in a similar and regular fashion. The response pattern suggested that, whatever the stimuli, these participants were giving an NPP solution. Discrimination scores were calculated, therefore, on the assumption that allergy responses would be made to elements and no allergy responses would be made to compounds. As a consequence negative discrimination would mean that participants were proposing a PPP solution to a problem.

Analysis was conducted with an Age (3) by Problem (2: A, B, AB (AB); C, D, CD (CD)) by Trial (5) ANOVA. This revealed a significant effect of Age ($F_{(2,73)} = 7.2$, $p<0.002$) and Trial ($F_{(4,292)} = 4.3$, $p<0.003$), but not of Problem, indicating that responses varied according to age and there had been some inconsistency in participants' responses, although there were few overall differences between the AB and CD problems. The introduction of AH4 as a covariate did not attenuate the significance in terms of Age ($F_{(2,68)} = 8.95$, $p<0.001$) or Trial ($F_{(4,272)} = 3.23$, $p<0.015$). Bonferroni post-hoc tests showed significant differences were confined to those between the Young and YO groups (Mean Difference = 0.403, SE= 0.11, p=0.001). There were significant interactions between Trial and Age ($F_{(8,292)} = 2.53$, $p<0.015$), Problem and Trial ($F_{(4,292)} = 4.03$, $p<0.0035$), and Age, Problem, and Trial ($F_{(8,292)} = 2.12$, $p<0.035$).

Figures 8.3.1 and 8.3.2 in conjunction with the statistical analyses go some way to explaining the pattern of these results. It can be observed that the Young group were almost completely consistent in their responses, and that regardless of the problem they gave a NPP response, showing that they were generalising the solution to the Stage 2 AB problem to their predictions for the CD problem. The relative inconsistency of older participants is reflected in the Age by Trial interaction and the Problem by Trial interaction can be interpreted as reflecting the uncertainty of the older groups. These interpretations are reinforced by the





Age by Trial by Problem interaction in that differentiation between the problems was confined to the older groups, particularly the YO.

Table 8.6: Summary Statistics for Figure 8.3.1

	Young		Young Old		Old Old	
Block	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.90	.37	.50	.67	.72	.51
2	.89	.40	.50	.72	.89	.22
3	.92	.26	.67	.60	.89	.22
4	.95	.30	.46	.85	.78	.44
5	.93	.33	.57	.74	.78	.44

Table 8.7: Summary Statistics for Figure 8.3.2

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.92	.34	.26	.75	.67	.50
2	.93	.33	.67	.58	1.00	.00
3	.91	.42	.61	.58	.78	.44
4	.93	.33	.43	.77	.78	.44
5	.92	.34	.50	.78	.78	.44

Further analysis sought to establish the extent to which participants' Stage 1 learning had been changed by acquiring Stage 2 contingencies, establish age related differences, and identify predictor variables. Recall that according to associative learning theories CD problem responses at test should be identical to those learned in Stage 1, whereas AB problem contingencies had been re-valued during Stage 2 and this difference should be preserved at test, unless participants were using a wholly elemental strategy. By this logic a reasonable measure of forgetting should be the difference between Stage 1 and Test Stage responses to the CD problems since this should have been preserved in the face of Stage 2 revaluation. To this end the average Test Stage CD responses were subtracted from the average Stage 1 final trial CD responses, to indicate the extent to which they had been changed over the course of Stage 2. This was calculated by taking the root of the squared difference between the average element predictions on the final trial of Stage 1 and average Test Stage element responses added to the root of the squared difference between the average compound predictions in the last trial of the Stage 1 and the Test Stage divided by two. This resulted in a number between 1 and 0 indicating the extent of change between Stage 1 and the Test Stage for compounds and elements of the CD problem, henceforward referred to simply as CD Change. Equation 8.1 illustrates this calculation, below, where C_{S1}

and D_{S1} indicate Stage 1 elements, CD_{S1} Stage 1 compounds, C_T and D_T Test stage elements and CD_T Test Stage compounds, and *n* the number of datum for each stimulus type.

Equation 8.1:

$$CDChange = \sqrt{\frac{\left(\left(\frac{\Sigma C_{S1} + \Sigma D_{S1}}{nC_{S1} + nD_{S1}}\right) - \left(\frac{\Sigma C_T + \Sigma D_T}{nC_T + nD_T}\right)\right)^2 + \left(\left(\frac{\Sigma CD_{S1}}{nCD_{S1}}\right) - \left(\frac{\Sigma CD_T}{nCD_T}\right)\right)^2}{2}}$$

Figure 8.4 shows the mean CD Change scores for each age group. Note that the pattern of differences reflects the earlier results. The Young group's CD Change scores were greatest and indicate an almost total interference of Stage 1 CD learning despite the fact that only the AB problem was re-valued. The OO group's CD Change was next greatest, followed by the YO group's which was least. As surmised before, this may reflect the OO group's relatively poor learning in Stage 1 and better Stage 2 accuracy relative to the YO group. A One Way ANOVA revealed significant age differences in CD Change scores ($F_{(2,73)} = 5.98$, $p<0.005$) that remained significant after the introduction of AH4 as a covariate ($F_{(2,68)} = 5.58$, $p<0.01$). Bonferroni post-hoc tests showed that this difference was confined to the Young and YO groups (Mean Difference = 0.196, S.E. = 0.58, $p<0.0035$).

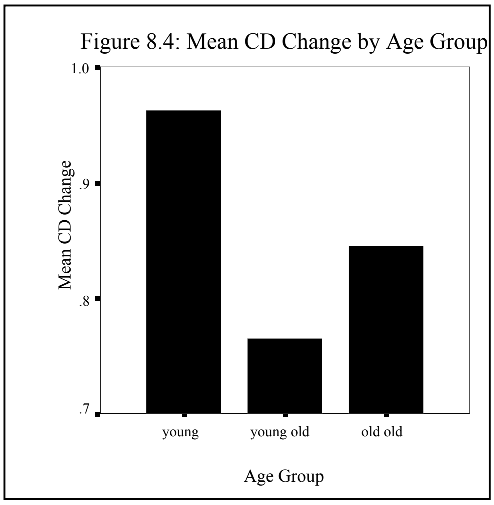


Table 8.8: Summary Statistics for Figure 8.4

Young		Young Old		Old Old	
Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
.96	.16	.77	.29	.84	.28

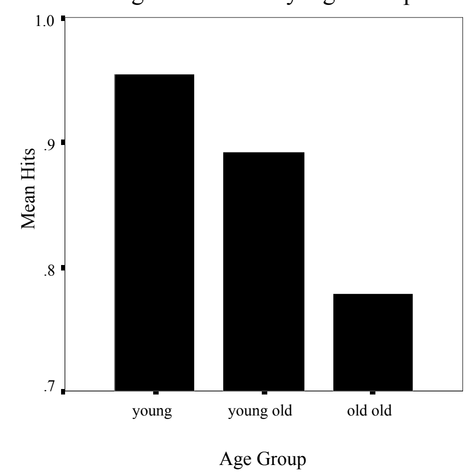
The next analysis was a multiple regression using the enter method with CD Change as a dependent variable. Predictor variables entered into a correlational analysis were Age, AH4 Total, DC, MHV, YE, McQ, and time taken to complete Stage 2 (S2 Time). The last predictor variable was entered since longer time intervals between Stage 1 and test may have caused participants to simply forget Stage 1 contingencies. Initial correlations revealed an association between CD Change and Age ($r = -0.34$, $p<0.0025$), AH4 Total ($r = 0.38$, $p<0.0045$), YE (0.39, $p<0.001$), and S2 Time ($r = 0.36$, $p<0.0025$). These four variables were therefore entered as predictor variables in a multiple regression analysis with CD Change as a dependent variable. This analysis revealed a significant model ($F_{(4,71)} = 8.15$, $p<0.001$, Adjusted $R^2 = 0.346$) with Age (Beta = -0.4, $p<0.0055$), AH4 Total (Beta = 0.37, $p<0.0025$), and YE (Beta = 0.47, $p<0.0035$) emerging as significant predictor variables. The failure of S2

Time to significantly predict interference suggests that this factor was not a confounding variable in the current experiment, and that forgetting of the CD problem contingencies were not systematically predicted by elapsed time.

8.1.6.2: Compound Recognition

Recall that participants were asked to state whether they had seen, during the course of the experiment, the three compounds used (AB, CD, and EF) and three novel compounds (AC, BE, CF) or not. Two measures were derived from these data: hits and false recognition, both expressed as a number between 0 and 1. Hits refers to the proportion of compounds out of six correctly identified as having been seen or unseen during the experiment whilst false recognition is calculated, following LaVoie et al. (2006), as the difference between the percentile proportion of hits minus the percentile proportion of false alarms taken away from one. This results in a number between 0 and 1 indicating the proportion of falsely recognised items after controlling for correct recognition.

Figure 8.5: Hits by Age Group



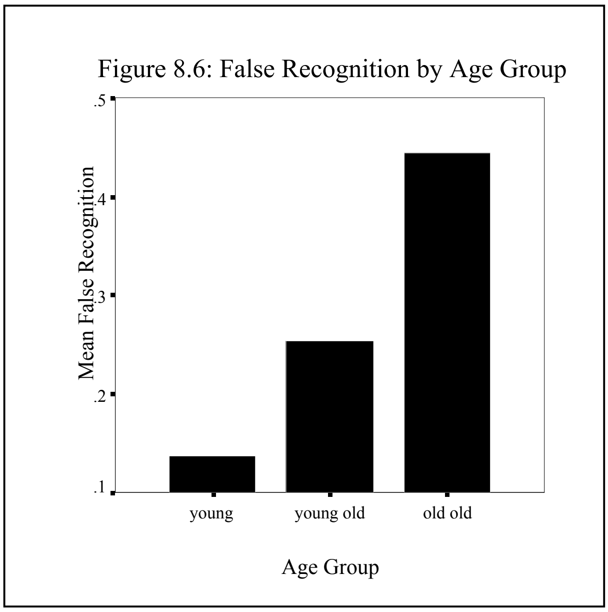


Table 8.9: Summary Statistics for Figures 8.5 & 8.6

	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Hits	.95	.09	.89	.13	.78	.17
False Recognition	.14	.27	.25	.34	.44	.33

The analysis itself was by two One Way ANOVAs: one for each dependent variable, but both with Age Group as a between subjects factor. The Hits analysis demonstrated a significant effect of Age Group ($F_{(2,73)} = 9.72$, $p<0.001$) that remained significant once AH4 had been entered as a covariate ($F_{(2,68)} = 4.73$, $p<0.15$), and Bonferroni post-hoc analysis showed that differences were significant only between the Young and OO groups (Mean Difference = 0.18, SE= 0.042, p=0.001; see Figure 8.5). The data for False Recognition followed a similar and statistically significant effect of Age Group ($F_{(2,73)} = 4.32$, $p<0.02$; see Figure 8.6). Bonferroni post-hoc tests again showed that this difference was, again, confined to Young versus OO groups (Mean Difference = 0.31, SE= 0.08, $p<0.02$). The effects of age on False Recognition were, however, eliminated by the introduction of AH4 as a covariate ($F_{(2,68)} = 1.94$, $p>0.05$), giving some credence to LaVoie's (2006) assertion that false recognition reflects higher order processing.

8.1.7: Experiment 9 Discussion

Stage 1 of this experiment derived results that were largely anticipated. There were age differences in the initial acquisition of the contingencies in that older groups learned more slowly than younger groups and less completely, although all groups discriminated significantly between elements and compounds by the final trial. Age predicted OA, but not FTA, suggesting that perhaps Age deficits were confined to the early part of the experiment, although the lack of an Age by Blocks interaction makes this less likely. YE was, on the other hand a predictor of both OA and FTA. This indicates that younger and more educated older participants may be better at learning. Perhaps the latter had kept more mentally active, giving support for the disuse hypothesis. This interpretation is consistent with the observation that Age differences were attenuated by covariance with AH4 scores in this stage. It may be that older, more educated people were better able to induce a simple rule to aid solution.

In Stage 2 all groups learned the AB problem more slowly than the EF problem, consistent with the predictions of unique-cue and configural theories, but the overall age effects were, surprisingly, confined to a difference between Y and YO, rather than OO, groups. Indeed the OO group learned better than the YO group on both problems, although not significantly so. On face value this suggests a curvilinear relationship between Age and vulnerability to proactive interference. An alternative interpretation of this may be that the YO had learned the Stage 1 contingencies better, or perhaps induced a threshold rule of summation that had helped with the PPP but was a hindrance in learning the NPP in Stage 2. This is also evidence of perseverative responding since the YO may have persevered with the PPP pattern of responses more than the OO because these responses were better learned and thus more difficult to change. This observation could therefore be seen as supporting a FL theory explanation of cognitive ageing. Perhaps this underlies the ability of AH4 scores to attenuate the main effect of Age and the Blocks by Problem interaction, The multiple regression analysis gave Age and AH4 Total as predictors, so it is possible that these proactive interference effects could be subsumed by a general fluid intelligence factor, perhaps reflecting the analogical reasoning and pattern recognition aspects of the AH4 test. On the other hand the best predictor of FTA was YE and again this may be interpreted as support for disuse theories, perhaps in limiting the extent of learning rather than learning rate, although there were no significant predictors of FTA for the EF problem. In the EF problem AH4 Total, but not Age, predicted OA, suggesting again that the ability to reason analogically and detect patterns and regularities helped learn the Stage 2 discriminations overall.

At Test one can assume that preservation of discriminations between elements and compounds reflects the use of configural assumptions whereas the inability to discriminate suggests the use of elemental strategies. In this sense the YO group seemed to revert to an elemental strategy, possibly as a result of their poor learning in Stage 2, although the OO did not. In another sense, however, Test Stage responses were even more unexpected than Stage 2 since it was the Young who had suffered the greatest change to Stage 1 CD responses, although this was clearly rule rather than associatively based and, as such, represents what one might term heuristic interference rather than the forgetting this measure may be interpreted as representing. Paradoxically it seems that it was the Young group's superiority at learning that seemed to cause the interference since they would have been more prone to inducing rules and generalising from them, and this may underlie the observation that age differences in CD Change were attenuated by AH4 scores. Young participants were almost totally consistent in their responses in the test stage, whereas older participants gave far less regular responses, suggesting rapid extinction, or unlearning, poorer retention of responses, and a possible increasing reliance on summation strategies. The Young also discriminated more between elements and compounds in both problems. In a continuation of their poor performance in Stage 2 the YO discriminated least and may reflect interference as a result of the disparity between Stage 1 and Stage 2 contingencies and a FL mediated inability to map stimuli to appropriate responses. The OO responses were more consistent and may reflect generalisation from their most recent associative learning in Stage 2 rather than rule application.

It is certainly the case that the extent of change to the unseen CD problem poses profound difficulties for the application of associative learning theories to HCL beyond predicting the acquisition of initial contingencies. Indeed it seems as though younger participants were, ultimately, taking more of a problem solving approach to the experiment than an associative learning approach. Age, AH4 Total, and YE predicted the extent of change between Stage 1 and Test Stage responses to the CD problem. The Age effect reminds us that although there were some differences between YO and OO the major differences were between young and old more broadly and suggest, again, that age may mediate rule induction, probably due to initial associative learning deficits. The predictive nature of AH4 Total may be related to the ability to spot patterns and regularities in patterns of stimulus-response associations, whereas the effect of YE may be due to better maintenance of cognitive ability, although one should note once again that these two variables predicted more CD Change, not less. In this instance FL mediated flexible, rule based thinking may, paradoxically, cause greater apparent forgetting in learning, or, more likely, heuristic interference in conditional learning. The inability of S2 Time to predict CD Change suggests that time between Stage 1 and Test was not a factor in the current experiment.

Overall this experiment provides a demonstration of the profound effects that rules can have on our responses and the limitations of the applicability of associative learning theory to HCL. Although an associative analysis is possible for Stages 1 and 2 the predictions of associative learning theories are almost redundant in terms of explaining Test data. There is also evidence to suggest age differences in the extent of heuristic interference in conditional learning, in that it had a more acute effect on the Young group's responses. The next experiment looks at these phenomena in more detail.

Despite being less prone to rule based interference older participants were less likely to correctly recognise compound stimuli as having been seen during the experiment. This time the biggest differences returned to the more familiar pattern of being significant between Young and OO groups. This indicates that at least some of the overgeneralisation as evidenced by poor discrimination between elements and compounds during the Test Stage may have been due to MTL mediated associative deficits, although the biggest differences in terms of responses at Test were between the Y and YO groups. Certainly, the observation that AH4 scores did not render age differences non-significant when entered as a covariate suggests that compound recognition may be independent of higher level cognitive abilities. Furthermore the significant age differences in false recognition suggest that while memory deficits existed they may not entirely underlie test stage responses in this experiment since false recognition is associated with a decline in executive, rather than purely mnemonic, abilities. Again, the observation that covariance with AH4 scores rendered age differences non-significant supports this interpretation. Overall this is a complex picture, and test responses seem to be more related to perserveration of learnt responses from Stage 2 and an heuristic, rule based overgeneralisation between stimuli rather than associative interference.

8.2: Experiment 10 Introduction

Experiment 9 produced some surprising results in that Stage 2 discriminations were more poorly learned by the YO rather than the OO group, perhaps because they had induced a partial rule in order to solve the Stage 1 PPPs and lacked the cognitive flexibility to reverse it. The Young group, on the other hand seemed able to transfer, by analogy, their learning of the easier PPP to the solution of the more difficult NPP. As a consequence this may be an instance of Easy-Hard transfer between associative discriminations following discrimination reversal (e.g. Suret et al. 2003) or, perhaps more likely, a case of applying an heuristic learned in an easy problem to a harder problem (e.g. Novick & Holyoak, 1991). The latter possibility may be more plausible since it was the Young group who had been most prone to retroactive interference in that they had generally applied the most recently learned heuristic to their Test Stage predictions. Given this assumption that analogical transfer of a solution had occurred from the easier PPP to the harder NPP it makes sense to suggest that reversing the order of presentation should alter the pattern of test stage responses, since there should be less transfer of a solution from Easy to Hard than from Hard to Easy. This leads to the prediction that proactive interference from Stage 1 to Stage 2 should be greater than in Experiment 9 in this instance. The change may, however, not affect the OO group's learning to the same extent since they seemed less likely to take advantage of the similarities between the problems to aid solution. Indeed one could expect the same basic pattern of results.

Formal quantitative predictions for Experiment 10 are largely similar to those derived from the models for Experiment 9. As far as the Rescorla-Wagner model is concerned they are identical, since elements are still equally likely to be associated with outcome or no outcome and would therefore each acquire an associative strength of 0.5λ, and compounds the sum of the value of their elements, 1λ, irrespective of whether they are NPPs or PPPs, so these simple predictions would be carried forward to Test. Adopting a threshold of activation of $\lambda > 0.5$ would, paradoxically, mean that a strictly elemental approach would lead to correct responses made to Stage 2 contingencies from Stage 1 learning, so in this sense a strictly elemental approach may lead to diminished, rather than increased, amount of proactive interference.

A unique-cue approach would predict in Stage 1 that compounds ABW- and CDX- would acquire unique cues W and X that would accrue an associative strength of -2λ to allow prediction of no response as elements A, B, C, and D would have a value of 1λ. In Stage 2 elements A, B, E, and F would lose associative strength and have an associative strength of 0λ, compound AB would acquire a new unique cue Y, and EF the unique cue Z. Here both Y and Z would accumulate an associative valence of 1λ. At Test the same problems occur as in

Experiment 9 as far as the predictions of unique-cue theory are concerned. It is unclear whether the AB compound would be treated as having either unique cue W or Y, and so could be worth either -2λ or 1λ. Responses to C, D, and CD should, however, be as per Stage 1 and elements A and B should have an associative strength of 0λ.

Pearce's model, however, makes the unequivocal prediction that Stage 1 elements would acquire strengths of 2λ and their compounds -2λ. In Stage 2 elements would accrue associative values of -1λ and their compounds 2λ. At Test A, B, and AB should remain unchanged from Stage 2 and C, D, and CD would attract responses consistent with Stage 1.

Experiment 10 was, therefore, essentially a replication, the only difference being that Stage 1 confronted participants with a NPP (A+, B+, AB-; C+, D+, CD-) and Stage 2 stimuli were arranged as a PPP (A-, B-, AB-; E-, F-, EF+).

8.2.1: Participants

Participants were 38 'Young' undergraduates (Mean Age = 24.04, S.D. = 7.34), 21 YO participants (Mean Age = 69.34, S.D. = 4.42), and 13 OO volunteers (Mean Age = 80.51, S.D. = 3.6).

Table 8.10: Participant Summary Statistics

	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Age	24.04	7.34	69.34	4.42	80.51	3.60
Years Education	14.00	.00	10.95	2.16	11.31	2.53
AH4 Total	78.66	16.78	65.43	15.35	55.18	24.49
Digit Cancellation	23.84	5.63	16.00	2.76	15.09	3.48
MHV	25.97	3.27	32.24	3.43	30.75	4.39
MacQuarrie Total	.	.	96.29	24.23	84.58	23.87

8.2.2: Design

Stimuli were the same as Experiment 9, as shown in Table 8.11. As stated previously the only differences between the present and previous experiment was that Stage 1 now contains two concurrent NPPs and Stage 2 two concurrent PPPs, including a revalued A, B, AB problem.

Table 8.11: Experiment 10 Design

Stage 1			***Stage 2***			***Test***		
	1	2		1	2		1	2
A+	Chocolate	Olive Oil	A-	Chocolate	Olive Oil	A	Chocolate	Olive Oil
B+	Fish	Bread	B-	Fish	Bread	B	Fish	Bread
AB-	Chocolate Fish	Olive Oil Bread	AB+	Chocolate Fish	Olive Oil Bread	AB	Chocolate Fish	Olive Oil Bread
C+	Olive Oil	Cheese	E-	Cheese	Chocolate	C	Olive Oil	Cheese
D+	Bread	Avocado	F-	Avocado	Fish	D	Bread	Avocado
CD-	Olive Oil Bread	Cheese Avocado	EF+	Cheese Avocado	Chocolate Fish	CD	Olive Oil Bread	Cheese Avocado

8.2.3: Procedures:

Procedures were identical to Experiment 9.

8.2.4: Results: Stage 1

8.2.4.1: Stage 1 Analysis

Here an Age by Blocks ANOVA demonstrated significant main effects of Blocks ($F_{(4,276)} = 44.69$, $p<0.001$) and Age ($F_{(2,69)} = 7.44$, $p<0.0015$). Bonferroni post hoc analysis showed that the Young's discriminations were significantly greater than the OO's (Mean Difference = 0.27, SE= 0.072, p=0.0015; see Figure 8.7). This is slightly different to Experiment 9 since the Bonferroni comparisons in that experiment were non significant, although similarly the main effect of Age was attenuated by AH4 as a covariate ($F_{(2,67)} = 0.862$, $p>0.05$). It is probable that the relative difficulty of the NPP led to greater age differences in Stage 1, and perhaps this difficulty was reflected by the elimination of any significant age differences by the introduction of AH4 as a covariate.

Final trial learning was again assessed using t-tests to check that participants were successfully discriminating between elements and compounds. Here all Young participants' predictions were all perfectly accurate and t was not calculated since there was no within group variance. Both the YO ($t = 10.12$, $df = 20$, $p<0.001$) and the OO groups ($t = 7.58$, $df = 12$, $p<0.001$) also successfully discriminated between the outcomes of elemental and compound stimuli and can therefore be said to have learned the problems.

Figure 8.7: Age by Blocks

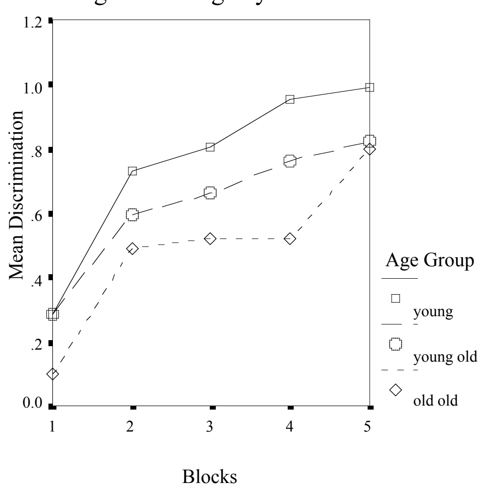


Table 8.12: Summary Statistics for Figure 8.7

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.29	.35	.29	.43	.10	.43
2	.73	.36	.60	.37	.49	.40
3	.81	.32	.66	.33	.52	.43
4	.95	.14	.76	.42	.52	.49
5	.99	.06	.82	.31	.80	.39

8.2.4.2: Stage 1 Multiple Regression

Initial correlations showed that OA was significantly associated with Age ($r = -0.27$, $p<0.025$) and AH4 Total ($r = 0.27$, $p<0.025$) but not Digit Cancellation (DC), Mill Hill Vocabulary Scale (MHV), MacQuarrie Total or YE. FTA, meanwhile, was significantly related to Age ($r = -0.32$, $p<0.0065$), AH4 Total ($r = 0.36$, $p<0.0035$), DC ($r = 0.33$, $p<0.0055$), and MHV ($r = -0.39$, $p<0.001$). The multiple regression model using the enter method for OA with Age and AH4 Total entered as predictor variables was not significant. The similar model for FTA with Age, AH4 Total, DC, and MHV as predictors, on the other hand, was significant overall ($F_{(4,64)} = 7.15$, $p<0.001$, Adjusted $R^2 = 0.266$). Analysis of individual predictors revealed that AH4 Total (Beta = 0.425, $p<0.0025$), Age (Beta = -0.4, $P<0.045$), and MHV (Beta = -0.545, $p<0.0015$) significantly predicted FTA scores. Note that MHV was significantly associated with Age ($r = 0.64$, $p<0.001$) and that the power of MHV as a negative predictor of FTA may be as a result of this relationship. Furthermore, the introduction of MHV as a covariate in an analysis of age differences in final trial discriminations resulted in no age differences ($F_{(2,67)} = 0.92$, $p>0.05$), whereas a simple one way ANOVA did ($F_{(2,69)} = 4.62$, $p<0.015$). This reflects the large correlation between MHV and Age, and in the absence of other instances where MHV has predicted accuracy or discrimination or theoretical reasons why crystallised intelligence should have any relationship with basic learning processes beyond its relationship with age it can be assumed that older participants in this experiment had particularly good vocabularies, but that this did not influence their learning ability.

8.2.5: Results: Stage 2

8.2.5.3: Stage 2 Analysis

Once more Stage 2 data were analysed using an Age (2) by Problem (2: AB & EF) by Blocks (5) ANOVA. This revealed significant main effects of Age ($F_{(2,69)} = 10.26$, $p<0.001$), Blocks ($F_{(4,276)} = 78.85$, $p<0.001$), and Problem ($F_{(1,69)} = 14.91$, $p<0.001$). Bonferroni post hoc analysis showed significant differences between the Young and both YO (Mean Difference = 0.32, S.E. = 0.074, $p<0.001$) and OO groups (Mean Difference = 0.23, S.E. = 0.088, $p<0.035$). Note that again the trend is for the YO performance to be worse than OO during Stage 2, although this was not significant. Furthermore, the effect of Age remained once AH4 had been entered as a covariate ($F_{(2,67)} = 5.51$, $p<0.007$), as did the effects of Blocks ($F_{(4,264)} = 10.25$, $p<0.001$) and Problem ($F_{(1,264)} = 11.103$, $p<0.002$). There were also significant Problem by Blocks ($F_{(4,276)} = 17.18$, $p<0.001$) and Age by Problem by Blocks ($F_{(8,276)} = 2.79$, $p<0.0065$) interactions. Figures 8.8.1 and 8.8.2 illustrate that AB problem discriminations are less accurate overall than EF problem discriminations, with older groups, particularly the YO, disadvantaged relative to the Young group. If anything the extent of proactive interference is more marked for this experiment than in Experiment 9. Although the Problem by Blocks interaction was attenuated by AH4 as a covariate ($F_{(4,264)} = 2.29$, $p>0.05$), the three way Age by Problem by Blocks interaction was not ($F_{(8,264)} = 2.56$, $p<0.015$).

Table 8.13: Summary Statistics for Figure 8.8.1

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
	.16	.50	-.20	.54	-.31	.58
2	.76	.40	.25	.73	.44	.61
3	.86	.29	.49	.54	.63	.39
4	.90	.24	.62	.54	.69	.40
5	.94	.20	.62	.53	.88	.19

Figure 8.8.1: Age by Blocks AB Problem

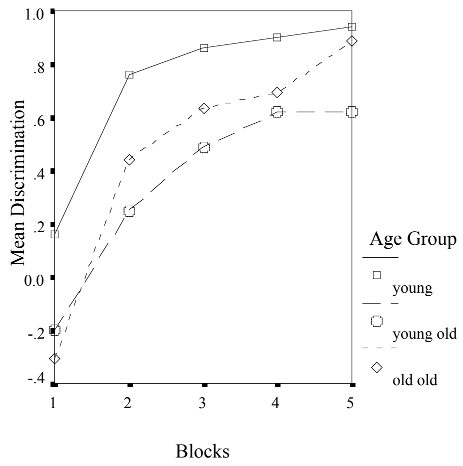


Figure 8.8.2: Age by Blocks EF Problem

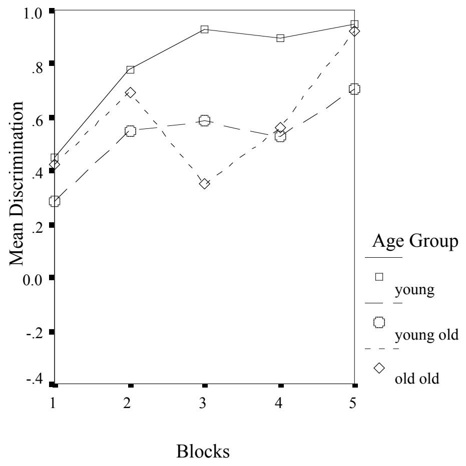


Table 8.14: Summary Statistics for Figure 8.8.2

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.45	.46	.29	.50	.42	.24
2	.78	.41	.55	.46	.69	.36
3	.93	.20	.58	.46	.35	.50
4	.89	.28	.52	.45	.56	.41
5	.95	.19	.70	.48	.92	.28

Again the extent to which participants successfully discriminated between Allergy and No Allergy outcomes by the final trial was examined with a series of t-tests. In this instance the Young group's responses to elements and compounds were significantly different for both the AB problem ($t = 43.33$, $df = 37$, $p<0.001$) and the EF problem ($t = 24.33$, $df = 37$, $p<0.001$). Similarly the OO group's responses to Allergy and No Allergy trials were significantly different for both problems (AB: $t = 17.73$, $df =12$, $p<0.001$; EF: $t = 12$, $df = 12$, $p<0.001$) as were the YO group's (AB: $t = 8.88$, $df =20$, $p<0.001$; EF: $t = 8.37$, $df = 20$, $p<0.001$).

8.2.5.4: Stage 2 Multiple Regression

Once again separate multiple regression analyses using the enter method were conducted for the AB and EF problems. In the AB problem OA correlated significantly with Age ($r = 0.413$, $p<0.001$), AH4 Total ($r = 0.551$, $p<0.001$), DC ($r = 0.329$, $p<0.0055$), and YE ($r = 0.603$, $p<0.001$) and FTA with AH4 Total ($r = 0.261$, $p<0.03$) and YE ($r = 0.326$, $p<0.0055$). For the EF problem OA correlated with Age ($r = 0.419$, $p<0.001$), AH4 Total ($r = 0.459$, $p<0.001$), DC ($r = 0.281$, $p<0.02$), and YE ($r = 0.514$, $p<0.001$) and none of the potential predictor variables were significantly associated with FTA.

In terms of the AB problem a regression model for OA with Age, AH4 Total, DC, and YE entered as predictor variables proved statistically significant ($F_{(4,65)} = 12.52$, $p<0.001$, Adjusted $R^2 = 0.4$) with AH4 Total (Beta = 0.293, $p<0.02$) and YE (Beta = 0.489, $p<0.002$) emerging as individually significant predictors. The model for FTA with AH4 Total and YE entered as predictors was also significant but predicted only a small proportion of the variance in FTA ($F_{(2,65)} = 4.48$, $p<0.02$, Adjusted $R^2 = 0.092$). Neither independent variable was a significant individual predictor of FTA, although YE approached significance (Beta = 0.275, $p = 0.056$). It may be that general intelligence only affected intial learning of the AB problem, particularly since AH4 scores only predicted overall accuracy here, and did not attenuate earlier age related differences and interactions when entered as a covariate for this stage.

For the EF problem the regression analysis with Age, YE, DC, and AH4 Total entered as predictors of OA provided a significant model ($F_{(4,65)} = 7.62$, $p<0.001$, Adjusted $R^2 = 0.277$). In this instance only YE proved a statistically significant predictor of OA (Beta = 0.336, $p<0.045$) although AH4 Total approached significance (Beta = 0.241, p = 0.069). Recollect that no potential predictor variables were associated with FTA for the EF problem, so no multiple regression analysis was conducted for this.

8.2.6: Test Stage Analysis

Recall that in Experiment 9 the Young group experienced almost complete rule based interference since their test stage responses to the CD problem had been re-valued such that they were the same as the AB problem that had been re-valued during Stage 2. This may have been the result of the application of a general heuristic. The following analysis will establish whether the same observation holds true for the current experiment.

8.2.6.1: Test Stage Responses and Consistency

In this instance discrimination scores were calculated on the assumption that participants will apply a PPP solution to Test Stage stimuli, since this was the pattern of responses learned in Stage 2. A negative discrimination score in this case will therefore indicate that participants are applying a NPP solution and scores near 0 that participants are inconsistent in their responses.

Analysis was once more begun with an Age (3) by Problem (2: AB; CD) by Trial (5) ANOVA. This showed no main effects of Age, Problem, or Blocks in this instance. There was, however, an interaction between Age and Problem ($F_{(2,272)} = 7.24$, $p<0.002$) that remained significant once AH4 scores had been entered as a covariate ($F_{(2,264)} = 3.83$, $p<0.025$). Figures 8.9.1 and 8.9.2 suggest that this interaction is due to inconsistencies in the way older participants approached the CD problem and that at least some, but by no means all, Young participants discriminated between the AB and CD problems.

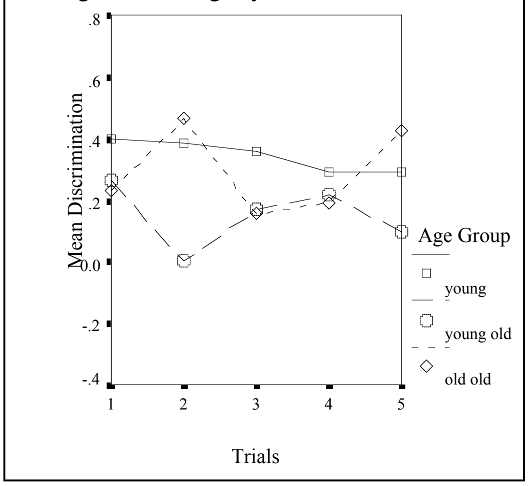


Figure 8.9.1: Age by Trials AB Problem

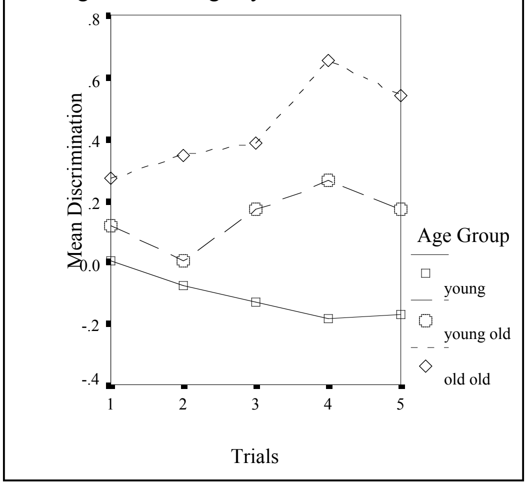


Figure 8.9.2: Age by Trials CD Problem

Table 8.15: Summary Statistics for Figure 8.9.1

	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.39	.82	.26	.85	.23	.75
2	.38	.87	.00	.81	.46	.59
3	.36	.91	.17	.80	.15	.85
4	.29	.95	.21	.82	.19	.83
5	.29	.93	.10	.77	.42	.61

Table 8.16: Summary Statistics for Figure 8.9.2

	Young		Young Old		Old Old	
Block	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.00	.90	.12	.77	.27	.70
2	-.08	.96	.00	.82	.35	.75
3	-.13	.95	.17	.78	.38	.74
4	-.18	.93	.26	.82	.65	.55
5	-.17	.96	.17	.90	.54	.63

The scarcity of observable Age, Problem, and Trial effects and interactions stands in stark contrast to the complex interactions seen in the last experiment. It seems that much of this may have been due to a lack of consistency in the Young group in terms of their predictions rather than across trials, as opposed to the overwhelming consensus encountered for this group in Experiment 9.

For the next round of analyses CD Change scores were first calculated in the same way as they were for Experiment 9 and again reflected the difference in allergy predictions for the CD problems between Stage 1 and the Test Stage. Figure 8.10 shows that the pattern of CD Change scores differed markedly from Experiment 9, with a more monotonic diminishing of the extent of change between Stage 1 and Test Stage responses in the current experiment. A One Way ANOVA, however, revealed no significant differences between age groups.

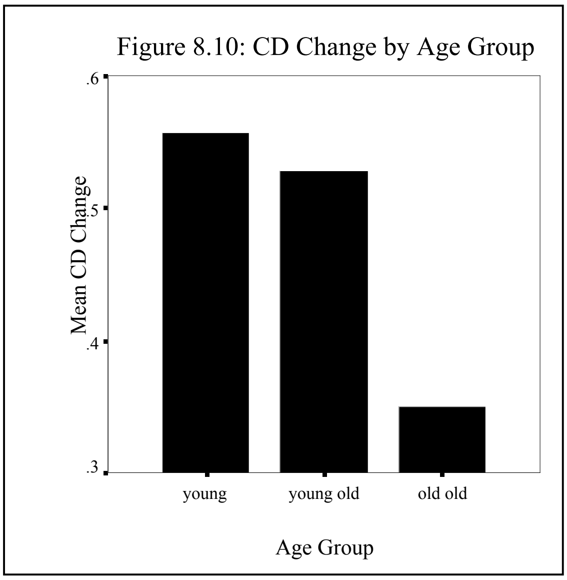


Table 8.17: Summary Statistics for Figure 8.10

Young		Young Old		Old Old	
Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
.56	.43	.53	.35	.35	.31

Once more predictor variables, Age, AH4 Total, DC, MHV, YE, McQ, and S2 Time, were entered into a correlational analysis with CD Change. This showed that interference was significantly associated with S2 Time ($r = 025$, $p<0.035$) but no other predictor variable. S2 Time was therefore entered into a regression analysis as a predictor of interference. In this instance S2 Time proved a significant but weak predictor of interference ($F_{(1,70)} = 4.73$, $p<0.035$, Adjusted $R^2 = 0.05$; Beta = 0.25, $p<0.035$). This implies that the longer participants spent on Stage 2 then the more interference they suffered regardless of age or any of the other individual difference variables. In fact it was the younger group that had spent longer completing Stage 2 ($F_{(2,69)} = 17.89$, $p<0.001$; Bonferroni Y vs. YO Mean Difference = 27.71s, $p<0.001$; Y vs. OO Mean Difference = 32.54s, $p<0.001$) This makes sense since one would expect CD Change to increase with S2 Time, although the amount of variance explained is small albeit consistent and there were no significant age differences in CD Change.

8.2.6.2: Compound Recognition

This task was identical to the previous experiment. Participants were given a list of six compounds (AB, CD, EF, AC, BE, CF) and asked to identify which they had seen and which they had not seen during the course of the experiment. The dependent variables again were numbers between 1 and 0 indicating the proportion of Hits and False Recognition.

Figure 8.11: Hits by Age Group

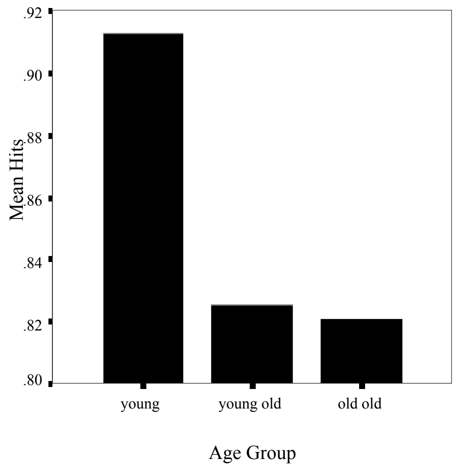


Figure 8.12: False Recognition by Age Grou

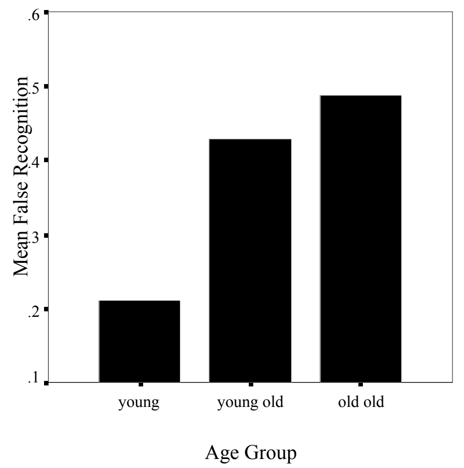


Table 8.18: Summary Statistics for Figures 8.11 & 8.12

	young		young old		old old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Hits	.91	.14	.83	.19	.82	.17
False Recognition	.21	.35	.43	.46	.49	.42

As before analysis was by two One Way ANOVAs: one for each dependent variable with Age group as a between subjects factor. In this case there were no significant age differences in number of Hits, but there was a significant effect of Age in terms of False Recognition ($F_{(2,69)} = 3.345$, $p<0.045$), although a Bonferroni Post Hoc analysis did not identify any specific differences between the three Age groups (see Figures 8.11 & 8.12; note the difference in scale), and the introduction of AH4 as a covariate rendered the age differences non-significant ($F_{(2,67)} = 2.24$, $p>0.05$), as in the last experiment.

8.2.7: Experiment 10 Discussion

Stage 1 results were largely as anticipated and very similar to those obtained from Experiment 9. The overall age differences again suggest that older groups' learning was worse than the younger group's, and in this instance there were significant differences between the Y group and both older groups. These observations may reflect the NPP's difficulty relative to PPPs, and the addition of AH4 as a covariate attenuated Age differences in Stage 1, suggesting that general cognitive ability may underlie age differences. Despite this, in the present experiment there were no significant predictors of OA, suggesting dissociation from other cognitive abilities. For FTA though Age, AH4 Total and MHV were all significant predictors. This may reflect age related deficits in rule induction due to an inability to recognise patterns and use this to solve problems. The significance of MHV as a predictor is somewhat problematic, but is likely to be related to Age since it is a strong correlate of Age ($r = 0.636$, $p<0.001$) and is likewise a negative predictor of FTA, and its relationship to age may explain why this factor attenuated age related differences when entered as a covariate.

Stage 2 results were also similar to those of Experiment 9 in that the trend persisted for the YO group to be the worst performers (see Figures 8.8.1 and 8.8.2). The overall Age difference was significant with the Young group again significantly better than the YO but also, in this case, significantly more accurate than the OO too, although there were no significant differences between the two older groups and all groups' discriminations were significant by the final trial. There was also a main effect of Problem as well as a Problem by Blocks interaction, demonstrating that proactive interference had occurred since participants had learnt the EF problem more quickly and more thoroughly than the AB problem. This implies that there was less transfer of learning from a hard to an easy problem than there had been from an easy to a hard problem, although the Age by Problem by Blocks interaction suggests that younger participants overcame proactive interference more quickly. Overall, though, it may be that the greater effort involved in encoding the Stage 1 NPPs may have resulted in a stronger, more persistent internal representation, less prone to change and therefore greater proactive interference. It also makes sense in terms of the earlier predictions made in terms of Hard-Easy transfers being more susceptible to proactive interference than Easy-Hard ones. Certainly, associative learning theories in general would either predict no differences in this sense between Experiments 9 and 10, since the same amount of relearning would have to be done regardless of which order the problems were presented in. In fact, a strictly elemental theory assuming an activation threshold of $\lambda > 0.5$ would predict that no learning would be needed in order to make correct responses to Stage 2 contingencies after Stage 1. It may be that the fact that PPPs are linearly soluble and participants did not need to engage configural processes could explain why AH4 covariance failed to render any of the observed differences non-significant, although this observation is difficult to account for.

For the AB problem AH4 Total and YE were significant predictors of OA. This was similar to Experiment 9 and may reflect participants' ability to detect patterns and regularities in the problem and change responses flexibly as appropriate. This seems at odds with the earlier observation that AH4 scores did not attenuate any of the main effects or interaction in the earlier analysis, but overall accuracy as a measure reflects all participants' ability to counter proactive interference rather than age differences *per se*. There were no significant predictors of FTA although the model including AH4 Total and YE as predictor variables was significant. For the EF problem, on the other hand, YE predicted OA and there were no predictors of FTA. This suggests that factors such as disuse and socio-economic status may mediate accuracy for this problem, which was clearly approached in a different way to the AB problem. Overall the results were similar to Experiment 9 for Stage 2 although there seems to have been greater pro-active interference, perhaps because participants, particularly younger ones, were less able to transfer their learning from a hard to an easy problem than from an easy to a hard problem.

The Test Stage results were more fundamentally different to those of Experiment 9. Here the Young group were not consistent in their predictions as they had been in the previous experiment. Indeed they did differentiate, to a point, between the AB and CD problems although not to the extent that would indicate a lack of heuristic interference. The lack of main effects and interactions compared to Experiment 9 suggests that there was little consensus in any groups about what should be predicted, although the attenuation of Age differences by AH4 covariance suggests that consistency may be determined by general cognitive ability rather than age as such. This stands in clear contrast to Experiment 9 to an extent that is surprising given the minor seeming manipulation of changing problem order, suggesting that Easy-Hard transfer is a robust empirical observation in HCL. Looking at the extent of change to the CD problem in the present experiment it is clear, again, that these results are very different. The lack of age differences here may reflect the overall inconsistency of responses to CD stimuli at test. Although S2 Time was a significant predictor of interference this was unlikely to have reflected an age related deficit since younger participants took longer over this stage on average, and Age was not even a correlate of CD Change in this instance.

Finally there were, again, age differences in the compound recognition task, although this time older groups were prone to false recognition but were not less likely to correctly recognise seen compounds. This suggests false recognition deficits associated with overgeneralisation between similar stimuli, and a decline in strategic, FL mediated processing rather than a mnemonic deficit, and this is reinforced by the observation that AH4 covariance accounted for the age related differences in false recognition. Although the effects of rule induction are of real interest and provide a major challenge to the applicability of associative learning theories to HCL problems there still remains the question of whether there are age differences in resistance to interference in non rule based conditional learning problems.

8.3: Experiment 11

Given that Experiments 9 and 10 had demonstrated the profound effect rule induction can have on responses to conditional learning problems the final experiment sought to investigate the phenomena of proactive and retroactive interference using a non-linear problem. This experiment is similar to the previous two in that it employs two learning stages and an unreinforced test stage. Each of the two learning stages is linearly soluble within itself, to allow all groups to learn them, but between stages an AB+, CD+ problem is re-valued as a AC-, BD- problem, thus rendering this part of the problem biconditional and non linear. This should cause proactive interference in Stage 2 and retroactive interference when Stage 1 contingencies are revisited in the test stage, which also asks participants to predict responses to two novel compounds, AD and BC. Stage 1 EF- and GH- contingencies are also featured in the test stage whereas Stage 2 IJ+ and KL+ were present to provide balance and prevent participants from merely learning that all stimuli in Stage 2 of the experiment resulted in No Allergy.

In terms of formal quantitative predictions the Rescorla-Wagner model would predict that participants would learn all Stage 1 and 2 contingencies, although IJ+ and KL+ would be learned more quickly than AC- and BD- since these stimuli would start the stage with an associative strength of 1λ whereas IJ and KL would begin at chance, 0.5λ. At test AB and CD would, by summation of their elements, have strengths of 0λ, as would AD and BC and, since their associative strength would remain unchanged, EF and GH would also have a value of 0λ.

The unique cue model might predict that in Stage 1 compounds AB, CD, EF, and GH would acquire cues S, T, U, and V, respectively, and that these cues would acquire all associative strength such that S and T would have a strength of 1λ and U and V a strength of 0λ. Alternatively one could point out that such cues are superfluous since the problem is linearly soluble and consequently unique cues would not form. Consequently, elements A, B, C, and D would all acquire a value of 0.5λ, and E, F, G, and H an associative strength of 0λ.

In the former case Stage 2 compounds AC, BD, IJ, and KL would acquire unique cues W, X, Y, and Z, respectively, and W and X would accrue a value of 0λ and Y and Z a strength of 1λ. Here the model would predict responses to AB and CD at Test to be either consistent with cues S and T at 1λ, or with cues W and X at 0λ. Responses to EF and GH would remain unchanged, and, since elements A, B, C, and D would have lost associative strength to the unique cues, AD and BC should both have a value of 0λ.

In the latter case A, B, C, and D elements would start Stage 2 with an associative strength of 0.5λ, so when compounds AC and BD form unique cues X and Y these should both acquire a value of –1λ to allow ACX and BDY to predict no response. IJ+ and KL+ should be easily learned and elements I, J, K, and L would accrue a strength of 0.5λ each. At Test AB, CD, AD, and BC should all have an associative strength of 1λ, through summation of the 0.5λ associative strengths of the elements A, B, C, and D. The EF and GH compounds' associative strengths should remain unchanged from Stage 1 at 0λ.

Pearce's (1987, 1994) configural model predicts that participants would learn Stage 1 contingencies with ease; compounds AB+ and CD+ would acquire associative strengths of 1λ and EF- and GH- values of 0λ. In Stage 2 the IJ+ and

KL+ compounds would be equally easy to learn, accruing strengths of 1λ. Note that the introduction of compounds AC- and BD- would effectively mean that participants would have to make a biconditional discrimination to preserve Stage 1 learning of AB+ and CD+. In these circumstances it is not unreasonable to make the same assumption concerning a reduction in the amount of associative strength generalised from perceptually similar stimuli as detailed at the beginning of Chapter 7. This assumption was as follows: 'when a problem is intractable because of perceptual overgeneralisation it is not unreasonable to suggest that solution may entail the engagement of higher cognitive processes in the form of inhibition of generalisation processes based on perceptual similarity'. Under these circumstances, or if generalisation processes led to a stimulus losing all of its associative strength, the assumption was made that generalisation of associative strength through perceptual similarity was halved. The basic Pearce (1987, 1994, 2002) model would predict that AC and BD would attenuate the generalised associative strength of AB+ and CD+ by gaining an associative strength of -1λ. That would mean that once this associative strength was generalised back to AB and CD at test they would lose all their associative strength at 0λ. Given that younger participants have been demonstrated to have a capacity to resist retroactive interference in learning (e.g. Shanks, Darby, and Azmi, 1998) it would not appear to be out of place to make the assumption that the amount of associative generalisation between AB, CD and AC, BD compounds be halved. Making this assumption means that during Stage 2 compounds IJ and KL would acquire an associative strength of 1λ, whereas AC and BD would lose associative strength to reach –0.5λ to counter generalisation from AB and CD since $V_{AC}/V_{BD} = -0.5 + (0.5 \cdot (0.5 \cdot 1)) + (0.5 \cdot (0.5 \cdot 1))$.

At Test, therefore, AB and CD should each generalise half of AC and BD's associative strengths of –0.5λ and have an associative value of 0.75λ since $V_{AB}/V_{CD} = 1 + (0.5 \cdot (0.5 \cdot -0.5)) + (0.5 \cdot (0.5 \cdot -0.5))$. Novel compounds AD and BC will have no associative strength of their own but would generalise associative value from AB, CD, AC, and BD. Here, if one assumes that the extent of generalisation through perceptual similarity is still halved AD and BC would be worth 0.125λ, as the following calculation demonstrates: V_{AD}/V_{BC} = 0 + (0.5•(0.5•0.75)) + (0.5• (0.5•0.75)) + (0.5• (0.5•-0.25)) + (0.5• (0.5•-0.25)). Alternatively, one might assume that since there is no objective need to suspend generalisation based on perceptual stimuli for novel compounds and this results in the very similar prediction that AD and BC would have associative strengths of 0.25λ since they would both generalise half the associative strengths of AC, BD, AB, and CD compounds V_{AD}/V_{BC} = 0 + (0.5•0.75) + (0.5•0.75) + (0.5•-0.25) + (0.5•-0.25).

In conclusion, the Rescorla-Wagner (1972) theory anticipates that compounds AB and CD would lose all their associative strength over the course of Stage 2 and would, in common with novel compounds AD and BC and also EF and GH, have an associative strength of 0λ at test. A unique cue modification would lead to no generalisation of associative strength between compounds and no revaluation of AB and CD during Stage 2, and makes equivocal predictions in terms of how novel compounds AD and BC would be responded to. Pearce's (1987, 1994, 2002) model, on the other hand, assumes that AB and CD would lose some associative strength through generalisation but that the discrimination between these and EF and GH would be preserved. It also predicts that allergy predictions in response to AB and CD compounds should be greater than that to AD and BC.

8.3.1: Participants

Participants for Experiment 11 were 29 Young undergraduates (Mean Age = 21.01, SD = 2.9), 19 YO volunteers (Mean Age = 69.47, SD = 3.85), and 13 OO contributors (Mean Age = 78.34, SD = 3.53).

Table 8.19: Participant Summary Statistics

	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Age	21.01	2.90	69.47	3.85	78.34	3.53
Years Education	14.10	.31	11.74	2.54	11.69	2.95
AH4 Total	82.20	12.62	67.24	18.31	58.78	22.82
Digit Cancellation	24.48	6.53	16.65	4.83	14.22	3.23
MHV	25.08	3.91	32.53	3.70	34.10	6.10
McQuarrie Total	.	.	98.67	25.47	92.10	21.65

8.3.2: Design

This experiment consisted of two learning stages and a test stage. Each learning stage consisted of compound stimuli presented in fifteen blocks of four trials and the Test Stage presented unreinforced compound stimuli in five blocks of six trials. Stage 1 required participants to learn an AB+, CD+, EF-, GH- discrimination and Stage 2 an AC-, BD-, IJ+, KL+ discrimination. The Test Stage presented participants with the Stage 1 AB, CD, EF, and GH stimuli along with two novel AD and BC compound stimuli. Table 8.20 summarises the design. After completing the experiment participants were asked to identify six compound stimuli that had been seen during the experiment (AB, CD, EF, GH, AD, BC) and six that had not been seen (AF, DG, BH, CE, EG, FG).

Table 8.20: Experiment 11 Design

Stage 1			Stage 2			Test		
	1	2		1	2		1	2
AB+	Olive Oil Avocado	Fish Chocolate	*C-*	Olive Oil Eggs	Fish Milk	AB	Olive Oil Avocado	Fish Chocolate
CD+	Eggs Potatoes	Milk Bread	BD-	Avocado Potatoes	Chocolat e Bread	CD	Eggs Potatoes	Milk Bread
EF-	Lettuce Chicken	Cheese Banana	IJ+	Bread Milk	Eggs Potatoes	EF	Lettuce Chicken	Cheese Banana
GH-	Cheese Fish	Olive Oil Avocado	KL+	Banana Chocolate	Chicken	GH	Cheese Fish	Olive Oil Avocado
						AD	Olive Oil Potatoes	Fish Bread
						BC	Avocado Eggs	Chocolate Milk

8.3.3: Procedures

Procedures were as detailed in Chapter 6. The compound recognition task was the same as for Experiments 9 and 10, although there were twice as many stimuli involved in the task.

8.3.4: Results: Stage 1

8.3.4.1: Stage 1 Analysis

Stage 1 analysis was carried out on discrimination scores for the AB+, CD+, EF-, GH- problem averaged out over five three trial blocks of stimuli using an Age (3) by Blocks (5) ANOVA. This resulted in main effects for Age ($F_{(2,58)}$ = 8.71, p<0.001) and Blocks ($F_{(4,232)}$ = 43.17, p<0.001) but no Age by Blocks interaction. Bonferroni post hoc analysis identified significant differences between the Young and YO (Mean Difference = 0.27, S.E. = 0.076, p<0.0025), and the Young and OO groups (Mean Difference = 0.28, S.E. = 0.086, p<0.0055), but no differences between the two older groups. Figure 8.13 (overleaf) illustrates this analysis and overall these data can be interpreted as showing that there is a simple age related decline in solving an AB+, CD+, EF-, GH- problem. As in previous experiments the introduction of AH4 as a covariate attenuated the effects of Age ($F_{(2,50)} = 2.02$, $p>0.05$). Further analysis on separate age group data using t-tests showed that the Young ($t = 57$, $df = 28$, $p<0.001$), YO ($t = 6.48$, $df = 18$, $p<0.001$), and OO groups ($t = 17.73$, $df = 12$, $p<0.001$) were significantly discriminating between Allergy and No Allergy trials and had therefore learned the problem

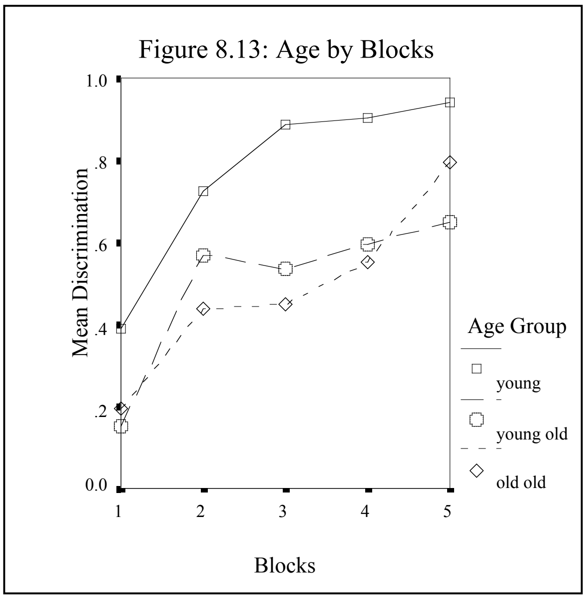


Table 8.21: Summary Statistics for Figure 8.13

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.39	.26	.15	.28	.19	.39
2	.72	.34	.57	.36	.44	.42
3	.89	.24	.54	.45	.45	.47
4	.90	.20	.60	.42	.55	.45
5	.94	.16	.65	.42	.79	.25

8.3.4.2: Stage 1 Multiple Regression

Initial correlations indicated that OA was significantly associated with Age ($r = -0.51$, $p<0.001$), AH4 Total ($r = 0.44$, $p<0.0015$), DC ($r = 0.31$, $p<0.03$), and YE ($r = 0.52$, $p<0.001$) therefore these variables were entered into a regression analysis using the enter method as predictor variables. This resulted in a significant model ($F_{(4,48)} = 5.996$, $p<0.0015$, Adjusted $R^2 = 0.29$) with YE emerging as the sole significant predictor of OA in stage 1 (Beta = 0.34, $p<0.05$).

In the FTA analysis Age ($r = -0.33$, $p<0.015$), AH4 Total ($r = 0.29$, $p<0.05$), DC ($r = 0.36$, $p<0.015$), and YE ($r = 0.495$, $p<0.001$) were significantly associated with the variable of interest and were entered as predictor variables. The model again proved significant ($F_{(4,48)} = 5.87$, $p<0.0015$, Adjusted $R^2 = 0.28$), with YE emerging yet again as the sole significant predictor of FTA (Beta = 0.54, $p<0.0025$). Since Age *per se* is not on its own a significant predictor in either analysis whereas YE is a significant predictor of both OA and FTA it is probably the case that younger and older better educated people did best in this stage of the experiment.

8.3.5: Results: Stage 2

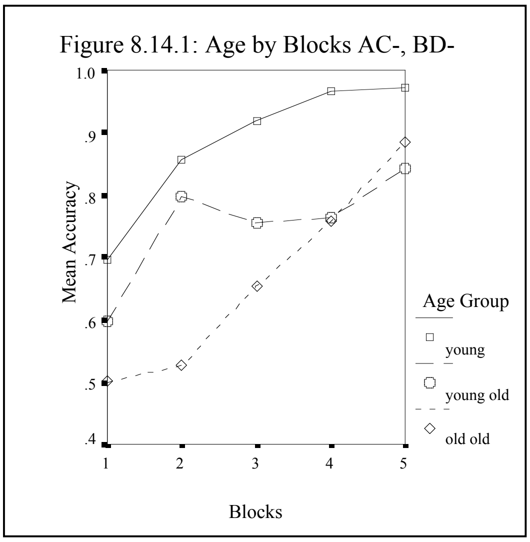
8.3.5.1: Stage 2 Analysis

Given that acquisition of the AC-, BD- contingencies should be more difficult than learning the novel IJ+, KL+ associations due to proactive interference this stage was analysed using accuracy scores. This allows accuracy for the two types of contingencies to be compared. Initial analysis was by an Age (3) by Problem (2: AC/BD and IJ/KL) by Blocks (5) ANOVA. This revealed significant main effects of Age ($F_{(2,58)} = 7.998$, $p<0.0015$), Blocks ($F_{(4,232)} = 41.31$, $p<0.001$), and Problem ($F_{(1,58)} = 11.45$, $p<0.0015$), showing that participants were learning the discriminations, but differently according to Problem and Age. Bonferroni post-hoc tests showed that the Young group were significantly more accurate in their predictions than the YO (Mean Difference =

0.17, S.E. = 0.042, $p<0.025$) and OO (Mean Difference = 0.17, S.E. = 0.047, $p<0.0025$) groups but there were no significant differences between the two older groups. The main effect of Age was attenuated by the introduction of AH4 as a covariate ($F_{(2,50)} = 0.313$, $p>0.05$). There was, furthermore, a three way interaction between Age, Problem, and Blocks ($F_{(8,232)} = 4.26$, $p<0.001$) that remained significant after AH4 had been entered as a covariate ($F_{(8,200)} = 2.35$, $p<0.02$). Figures 8.14.1 and 8.14.2 illustrate this interaction and suggest that the OO group were poor at solving the AC-, BD- problem in the early stages of the experiment relative to other Age groups and suggesting they were more prone to proactive interference, although their performance improved towards the end of this stage. The YO, on the other hand, had started to learn the AC-, BD- discriminations almost as well as the Young but their accuracy decreased markedly after the second block of trials. In order to confirm this interpretation two Age by Blocks ANOVAs were performed separately for each problem. This showed that for the IJ+, KL+ problem there was a significant effect of Age ($F_{(2,58)} = 4.41$, $p<0.02$) and Blocks ($F_{(4,232)} = 28.87$, $p<0.001$) but no interaction, consistent with a monotonic age related decline. Bonferroni post-hoc tests also showed that the Age differences were solely between Y and OO groups (Mean Difference = 0.13, S.E. = 0.05, $p<0.04$). For the AC-, BD- predictions there were also main effects of Age ($F_{(2,58)} = 9.895$, $p<0.001$) and Blocks ($F_{(4,232)} = 30.78$, $p<0.001$) but also an interaction between Age and Blocks ($F_{(8,232)} = 2.99$, $p<0.0035$). Furthermore Bonferroni post-hoc tests demonstrated that age differences were significant between the Young and both OO (Mean Difference = 0.22, S.E. = 0.052, $p<0.001$) and YO (Mean Difference = 0.13, S.E. = 0.046, $p<0.02$) groups. This demonstrates that proactive interference from Stage 1 had differentially affected the older groups' ability to master the AC-, BD- discriminations relative to the novel IJ+, KL+ associations.

Table 8.22: Summary Statistics for Figure 8.14.1

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.70	.16	.60	.22	.50	.20
2	.86	.23	.80	.20	.53	.24
3	.92	.18	.75	.31	.65	.22
4	.97	.09	.76	.29	.76	.24
5	.97	.11	.84	.23	.88	.18



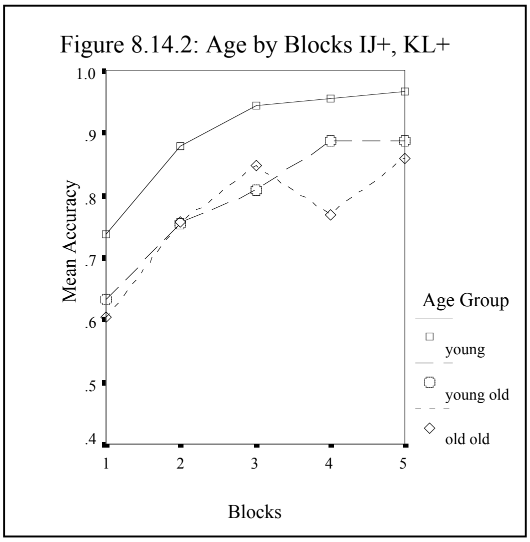


Table 8.23: Summary Statistics for Figure 8.14.2

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.74	.19	.63	.15	.60	.28
2	.88	.20	.75	.22	.76	.32
3	.94	.13	.81	.23	.85	.19
4	.95	.13	.89	.19	.77	.23
5	.97	.10	.89	.23	.86	.18

To test whether participants had ultimately learned the contingencies t-tests were performed on final trial data for each age group. This analysis showed that the Young group successfully discriminated between AC/BD and IJ/KL stimuli during the final trial ($t = 57$, $df = 28$, $p<0.001$), as did the YO group ($t = 7.39$, $df = 18$, $p<0.001$), and the OO group ($t = 25$, $df = 12$, $p<0.001$.

8.3.5.2: Stage 2 Multiple Regression

Given the significant main effect of stimulus response accuracies in this stage the stimulus types (AC/BD and IJ/KL) were separated for multiple regression analysis using the enter method. For AC/BD stimuli there were significant correlations between OA and Age ($r = -0.51$, $p<0.001$), AH4 Total ($r = 0.56$, $p<0.001$), DC ($r = 0.39$, $p<0.005$), and YE ($r = 0.322$, $p<0.015$) and these variables were entered as predictors for this analysis. This model proved a significant predictor of OA ($F_{(4,48)} = 8.46$, $p<0.001$, Adjusted $R^2 = 0.37$) with Age (Beta = -0.44, $p<0.0075$) and AH4 Total (Beta = 0.47, $p<0.0035$) emerging as significant individual predictor variables. For FTA response accuracy for AC/BD stimuli correlated with Age ($r = -0.29$, $p<0.025$), AH4 Total ($r = 0.46$, $p<0.0015$), and YE ($r = 0.27$, $p<0.035$). The regression model with these variables as predictors was significant ($F_{(3,49)} = 4.44$, $p<0.0085$, Adjusted $R^2 = 0.17$) and AH4 Total emerged as the sole significant individual predictor of FTA for AC/BD stimuli.

For the IJ/KL overall response accuracy was significantly associated with Age ($r = -0.373$, $p<0.0035$), AH4 Total ($r = 0.32$, $p<0.025$), and DC ($r = 0.32$, $p<0.025$). When these variables were entered into a multiple regression analysis using the enter method the model proved significant ($F_{(3,49)} = 4.15$, $p<0.015$, Adjusted $R^2 = 0.16$) and Age was revealed as the only individually significant predictor of OA for IJ/KL stimuli (Beta = -0.376, $p<0.04$). FTA for the IJ/KL stimuli was significantly associated with YE ($r = 0.28$, $p<0.035$) and the model derived from this predictor variable proved significant, although it only predicted a small amount of FTA variance ($F_{(1,59)} = 4.91$, $p<0.035$, Adjusted $R^2 = 0.061$; Beta = 0.277, $p<0.035$).

8.3.6: Test Stage Analysis

The fundamental questions to be answered by this analysis are whether participants preserved the Stage 1 AB+, CD+, EF-, GH- discriminations and whether their responses to AB and CD stimuli were different from their predictions for the novel AD and BC stimuli. Recall that the Rescorla-Wagner (1972) theory predicted no difference between responses to any of these stimuli since all elements would have acquired an associative strength of 0λ. Unique-cue theories predicted no differences between AB/CD and AD/BC compounds since both types of response would be reached by summation of elements A, B, C, and D. Predictions concerning differences between AB/CD and EF/GH were equivocal with unique cue theory, but suggest either strong or no discrimination. Pearce's (1987, 1994, 2002) configural theory predicts that the discrimination between AB/CD and EF/GH should be preserved and that responses to AB/CD should be significantly stronger than those given to AD/BC, so this constitutes a critical comparison. To this end two separate analyses were performed on these data: one comparing responses to AB/CD and EF/GH stimuli and another to compare predictions for AB/CD and AD/BC stimuli. In addition the compound recognition task in this experiment constitutes a sterner test of participants' stimulus-stimulus learning ability than the analogous tasks in Experiments 9 and 10 since there are 12 rather than 6 compounds involved.

8.3.6.1: Test Stage Responses AB/CD and EF/GH Contingencies

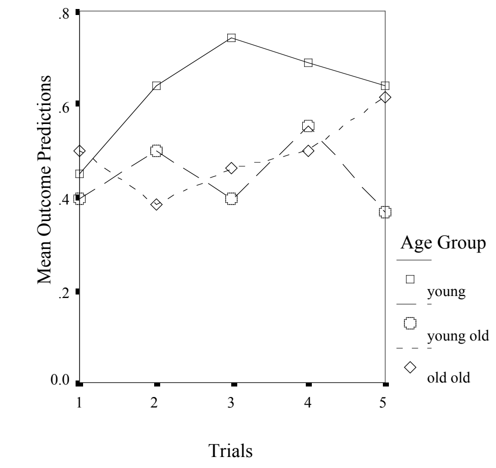


Figure 8.15.1: Age by Blocks AB/CD Stimuli

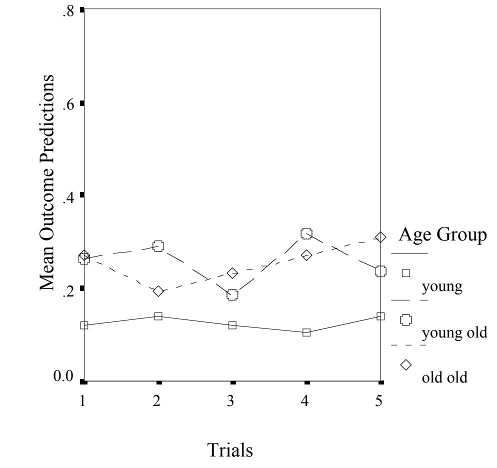


Figure 8.15.2: Age by Blocks EF/GH Stimuli

Table 8.24: Summary Statistics for Figure 8.15.1

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.45	.31	.39	.36	.50	.35
2	.64	.35	.50	.17	.38	.42
3	.74	.32	.39	.39	.46	.38
4	.69	.36	.55	.37	.50	.41
5	.64	.38	.37	.37	.62	.36

Table 8.25: Summary Statistics for Figure 8.15.2

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.12	.22	.26	.26	.27	.39
2	.14	.26	.29	.42	.19	.38
3	.12	.22	.18	.25	.23	.39
4	.10	.21	.32	.25	.27	.39
5	.14	.26	.24	.35	.31	.38

Comparisons between AB/CD and EF/GH stimuli were made with an Age (3) by Stimulus (2) by Trials (5) ANOVA. The main effect of Stimulus demonstrates that overall participants differentiated between the two stimulus types ($F_{(1,232)} = 29.51$, $p<0.001$), although the main effects of Age and Trials proved non-significant, suggesting that overall participants were consistent in their responses to the stimuli with no overall age differences, and this main effect was attenuated by covariance with AH4 ($F_{(1,200)} = 3.7$, $p>0.05$). There were, however, significant Stimulus by Age ($F_{(2,58)} = 3.92$, $p<0.03$) and Trials by Age ($F_{(8,232)} = 2.596$, $p<0.015$) interactions. In conjunction with Figures 8.15.1 and 8.15.2 this implies that the Young group preserved the Stage 1 discrimination best, and are therefore least susceptible to retroactive interference. It also suggests that younger participants were more consistent in their responses to unreinforced stimuli. Although this trend appears more marked for the EF/GH stimuli there was no Age by Stimulus by Trials interaction so this interpretation lacks empirical support. The Stimulus by Age interaction was rendered non-significant by entering AH4 as a covariate ($F_{(4,200)} = 0.78$, $p>0.05$), but not the Trials by Age interaction ($F_{(4,200)} = 2.18$, $p<0.035$). Further analysis was conducted using t-tests to determine whether each age group had consistently discriminated between AB/CD and EF/GH stimuli over the whole Test Stage. This showed significant differences in the Young ($t = 6.87$, $df = 28$, $p<0.001$) and YO ($t = 2.24$, $df = 18$, $p<0.04$), but not the OO group's responses to AB/CD and EF/GH stimuli. This can be interpreted as demonstrating that the OO had suffered greater interference to AB+, CD+ contingencies as a result of Stage 2 exposure to AC-, BD- associations.

In order to establish the extent of any interference a measure was calculated based on the differences in terms of AB and CD predictions between the final trial of Stage 1 and average test trial predictions. Figure 8.16 (overleaf) shows mean interference scores by age groups and illustrates that in this experiment interference seemed to increase with age, although a One Way ANOVA did not identify any significant differences between age groups.

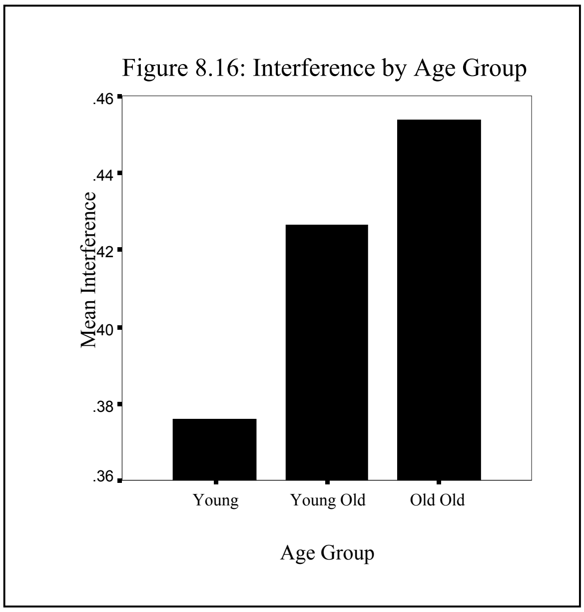


Table 8.26: Summary Statistics for Figure 8.16

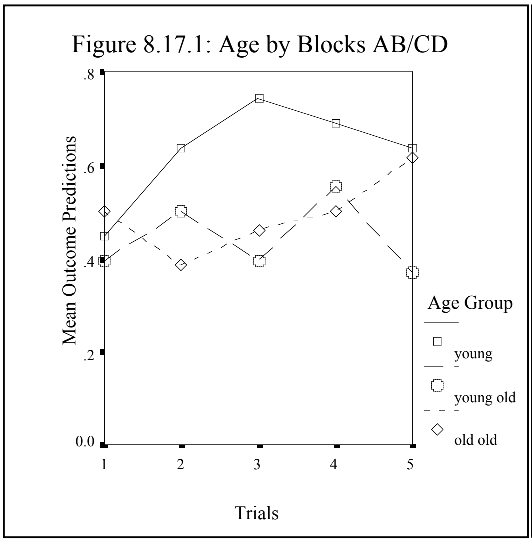
Young		Young Old		Old Old	
Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
.38	.25	.43	.22	.45	.33

Finally a multiple regression analysis using the enter method was performed with interference as a dependent variable and Age, AH4 Total, DC, MHV, YE, McQ, and S2 Time as potential predictors. Initial correlations revealed that only AH4 Total scores were associated with interference (r = -0.28, $p<0.05$) and that this variable was a significant but weak predictor of interference ($F_{(1,49)} = 4.19$, $p<0.05$, Adjusted $R^2 = -0.06$; Beta = -0.281, $p<0.05$).

8.3.6.2: Test Stage Responses AB/CD and AD/BC Contingencies

The next round of comparisons looks for similarities and differences between participants' responses to AB/CD relative to the novel AD/BC compounds. This should give an indication of how much participants are generalising between similar stimuli since all the constituent elements of the AD/BC compounds had been associated with both Allergy and No Allergy outcomes during the experiment, but AB/CD compounds had only, as unique configurations, been associated with Allergy outcomes.

The initial analysis was, again, by an Age (3) by Stimulus (2) by Trials (5) ANOVA. Once more there was no main effect of Age but there were significant effects of Stimulus ($F_{(1,58)} = 4.297$, $p<0.045$) and Trials ($F_{(4,232)} = 5.68$, $p<0.001$) as well as an interaction between Stimulus and Age ($F_{(2,232)} = 5.904$, $p<0.0055$). The interaction between Stimulus and Age remained significant once AH4 scores had been entered as a covariate ($F_{(2,200)} = 4.82$, $p<0.015$. An inspection of Figures 8.17.1 and 8.17.2 (overleaf) shows that participants were relatively inconsistent in their responses to these stimuli over the course of the experiment. Younger participants seemed to be less consistent in their responses to AB and CD stimuli relative to AD and BC stimuli, however, although they differentiated between stimulus types more than older groups.



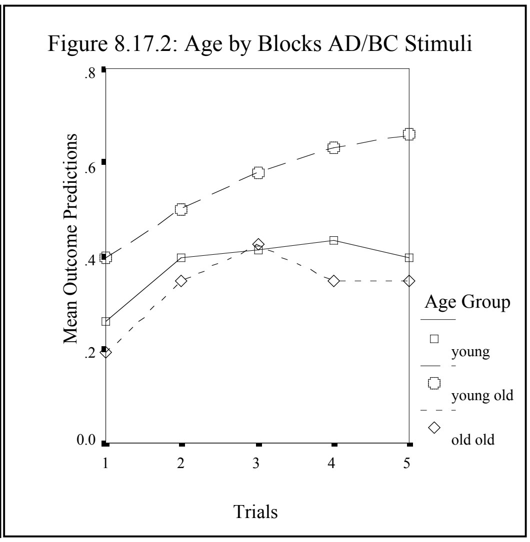


Table 8.27: Summary Statistics for Figure 8.17.1

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.45	.31	.39	.36	.50	.35
2	.64	.35	.50	.17	.38	.42
3	.74	.32	.39	.39	.46	.38
4	.69	.36	.55	.37	.50	.41
5	.64	.38	.37	.37	.62	.36

Table 8.28: Summary Statistics for Figure 8.17.2

Block	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
1	.26	.37	.39	.36	.19	.25
2	.40	.43	.50	.37	.35	.32
3	.41	.36	.58	.30	.42	.40
4	.43	.39	.63	.33	.35	.47
5	.40	.41	.66	.24	.35	.43

Further t-tests sought to ascertain whether participants consistently discriminated between these stimulus types over the whole test stage by comparing average responses to AB/CD and AD/BC stimuli in each Age group. This analysis showed that the Young group's responses to these stimuli were significantly different ($t = 3.19$, $df = 28$, $p<0.005$), as were the OO group's ($t = 2.77$, $df = 12$, $p<0.02$), but not the YO group's.

8.3.6.3: Compound Recognition Task

In this experiment participants were, once again, shown a list of compound stimuli made up of elements they had encountered during the experiment and asked to identify those that they had seen and those they had not seen. Seen compounds were AB, CD, EF, GH, AD, and BC whereas unseen compounds were AF, DG, BH, CE, EG, and FG. Scores for hits and false recognition were calculated as in previous experiments.

Analysis of Hits data by One Way ANOVA revealed a significant effect of Age ($F_{(2,58)} = 6.75$, $p<0.0025$) and Bonferroni post-hoc tests identified significant differences between Young and YO participants (Mean Difference = 0.11, S.E. = 0.04, $p<0.03$) and between Young and OO participants (Mean Difference = 0.15, S.E. = 0.05, $p<0.0055$), and that this age difference was attenuated by AH4 as a covariate ($F_{(2,51)} = 2.14$, $p>0.05$). An inspection of Figure

Figure 8.18: Hits by Age Group

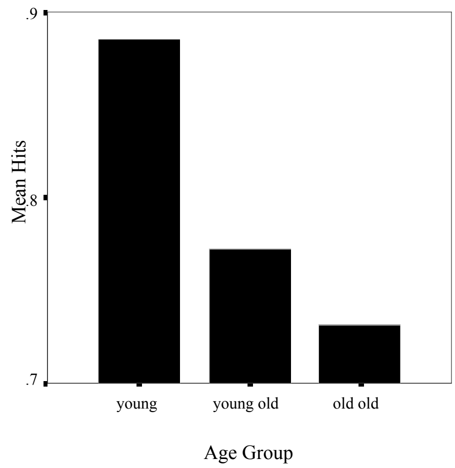
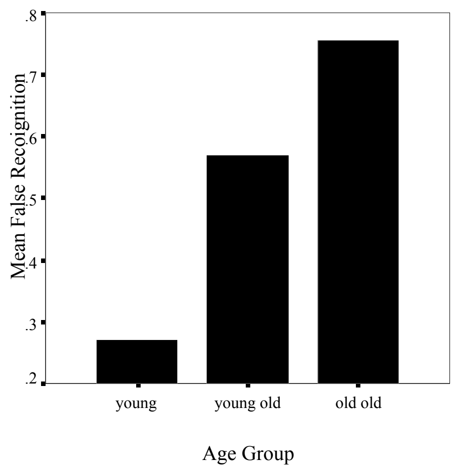


Figure 8.19: False Recognition by Age Group



8.18 confirms that younger people were more accurate in identifying which compounds they had seen and which they had not during the experiment.

Table 8.29: Summary Statistics for Figures 8.18 & 8.19

	Young		Young Old		Old Old	
	Mean	Std Deviation	Mean	Std Deviation	Mean	Std Deviation
Hits	.89	.11	.77	.15	.73	.18
False Recognition	.27	.28	.57	.36	.76	.54

A One Way ANOVA performed on False Recognition data again showed a significant effect of Age Group ($F_{(2,58)} = 8.704$, $p<0.001$) that was rendered non-significant once AH4 had been entered as a covariate ($F_{(2,50)} = 2.32$, $p>0.05$).

Bonferroni post-hoc tests showed that the Young group were significantly more accurate in their predictions than the YO (Mean Difference = 0.3, S.E. = 0.11, $p<0.025$) and OO (Mean Difference = 0.49, S.E. = 0.12, $p<0.001$) groups but there were no significant differences between the two older groups. Figure 8.19 (note the difference in scale) shows that younger participants suffered far less false recognition.

8.4: Experiment 11 Discussion

Stage 1 results were unsurprising and merely point to an age related decline in the ability to discriminate between compound stimuli in terms of different outcomes. AH4 scores entered as a covariate attenuated age differences, suggesting that they may be due to declines in general intelligence. In this instance YE again proved to be a significant predictor of both FTA and OA, suggesting that compound discrimination may be mediated by continuing intellectual activity or socio-economic status.

Stage 2 results were more interesting and point, again, to an age related vulnerability to pro-active interference since acquisition of AC-, BD- contingencies suffered relative to IJ+, KL+ associations for older groups but not for younger participants. Presumably this was due to perseveration through a deficiency in FL mediated inhibitory processes, and consequent overgeneralisation between stimuli, although all groups ultimately mastered the discriminations. Once again AH4 Total seemed to be a factor in resisting proactive interference since it predicted both OA and FTA for the AC-, BD- contingencies, whereas Age itself was a significant predictor of OA. Furthermore, AH4 scores entered as a covariate attenuated the main effects of Age, Blocks, and Problem, reinforcing the conclusion that fluid intelligence is implicated more broadly in resisiting interference and the engagement of configural learning processes. Conversely, the interaction of Age, Blocks, and Problem was not rendered non significant by covariance with AH4 scores, suggesting that some parts of the learning process, presumably basic associative processes, may not be determined by general cognitive ability. In all the last three experiments AH4 Total has been a predictor of accuracy in the re-valued Stage 2 variables, so perhaps resistance to proactive interference in HCL problems can be subsumed within a general fluid intelligence factor, even if associative learning ability *per se* cannot. The fact that Age predicted OA but not FTA in both problems may reflect the older groups' relatively poor start in learning the contingencies. For the IJ+, KL+ contingencies YE was a predictor, suggesting that these were mastered in a similar way to the Stage 1 discriminations.

The Test Stage analyses indicate that older participants were also more prone to retroactive interference since they did not preserve the discrimination between AB+, CD+ and EF-, GH- stimuli as well as the Young group, as indicated by the Stimulus by Age interaction. There were no age differences in the extent to which Stage 1 AB+, CD+ contingencies had been interfered with but the OO group did not significantly discriminate between these and the EF-, GH- contingencies, implying that the over seventy fives are significantly prone to interference. Given the evidence of previous experiments it may be that this indicates an elemental approach to learning, although this comparison alone does not rule out the use of a configural strategy. In this instance AH4 Total scores were the only individual predictors of interference, again a common factor with Experiment 9 and implying that the extent of interference may be due to a lack of cognitive flexibility and a declining ability to detect patterns in stimuli. Note that this would be exactly the kind of flexibility necessary to reduce the amount of associative strength generalised from other stimuli on the basis of perceptual similarity. Overall, though, the other groups resisted retroactive interference to an extent incompatible with basic elemental theories, suggesting either a unique-cue or configural approach, and the OO's inability to preserve the discrimination suggests they may have used an elemental strategy to predict test stage responses. This interpretation is reinforced by the observations that the Stimulus by Age interaction for ABCD and EFGH problems was attenuated when AH4 was included as a covariate. That the Age by Trials interaction was not rendered non significant by AH4 covariance suggests that some of the observed age related differences may be simple forgetting, but that higher level cognitive processes are required to preserve learning in the face of interference.

The assumption that the OO were using an elemental strategy to predict responses to stimuli is challenged, however, by the observation that they consistently discriminated between AB+, CD+ and the novel AD and BC stimuli. This suggests that the OO are using configural assumptions to predict responses to novel stimuli, and this may mean that they were unable to change their strategies between remembering existing discriminations and extrapolating from these to generate novel predictions. On closer inspection, however, they may have been using summation. Given that test responses to AB and CD were around chance levels (Mean = 0.49) for this group, indicating uncertainty, and compounds AC and BD elicited no allergy predictions from this group a process of summation would suggest that the predictions for compounds AD and BC would fall somewhere in between those values, at around 0.25, not far from the observed mean of 0.33. This does, of course, indicate some use of configural strategies and an ability to differentiate between different compounds composed of the same elements, but in a rudimentary way, and it is clear that responses to AB and CD are so close to chance levels they indicate guessing, rather than the adoption of any strategy. This interpretation is consistent with the observation that the Stimulus by Age remained significant after AH4 had been added as a covariate: older groups seemed not to be using sophisticated configural learning processes to extrapolate from existing learning and therefore did not need to engage higher order processing to this end.

In contrast the Young group seemed able to change their configural strategies to fit circumstance, and results indicate their ability to inhibit generalisation based on perceptual similarity in order to preserve existing learning and resist retroactive interference. In any event these data confirm the suspicions of Shanks, Darby, and Charles (1998) that the default human learning strategy is configural. In this case it was the YO whose responses indicated the most overgeneralisation since they failed to discriminate between AB/CD and AD/BC stimuli over the test stage, indicating that they may have been relying on a unique-cue strategy to both preserve the distinction between AB/CD and EF/GH contingencies and generate predictions for AD/BC stimuli. It is, therefore, entirely possible that the overgeneralisation between stimuli observed for older age groups may reflect an inability to change learning strategies to suit the occasion. This lack of cognitive flexibility is certainly consistent with the observation that fluid intelligence, as measured by AH4 scores, was a good predictor of resistance to proactive interference in Experiments 9, 10, and 11, and a good predictor of resistance to retroactive interference in the present experiment.

It could, further, be surmised that the interference effects may be the result of declining executive abilities, and there are some suggestions that this was the case in that older participants suffered more pro- and retro-active interference than young participants and were unable to change their learning strategy as appropriate. On the other hand the OO group's inability to preserve the discrimination between AB/CD and EF/GH contingencies may have been due to simple forgetting or the use of the summation strategy described earlier. In support of this interpretation there was progressive age related deterioration in both compound recognition and interference to Stage 1 AB+ and CD+ contingencies. On the other hand age differences in Hits were rendered non significant once AH4 covariance had been accounted for. It may be that simple recognition was affected by general cognitive ability here since there were twice as many compounds involved in the task as previous experiments, and strategic retrieval processes were engaged. In addition, older groups were more vulnerable to False Recognition, suggesting a more strategic processing deficit, and again these were attenuated by AH4 covariance. Overall, as far as the present investigation can tell, it seems plausible to assume that it is a combination of overgeneralisation and simple forgetting that contributes to greater learning interference as age increases. Chapter 9 will put the observations of the previous three chapters into the context of the theoretical framework outlined in the literature review.

Chapter 9: Conclusions and Discussion

9.1: Summary of Results

The research reported here has examined age related changes in human conditional learning (HCL), the extent and nature of pro- and retro-active interference in learning and the effects of rule and feature based generalisation on participants' responses to stimuli. Experiments One to Five looked at positive (PPP, e.g. A-, B-, AB+) and negative (NPP, e.g. C+, D+, CD-) patterning problems, differences between element and compound learning, more and less non-linear problems, and rule induction. These experiments also examined the effects of problem complexity in terms of number of responses available, number of exemplars or stimuli, and problem type. Experiments Six to Eight used conditional (e.g. AB+, AC+, DB-, DC-) and biconditional (e.g. AB+, CD+, AC-, BD-) problems to look further into the effects of problem linearity on age related learning deficits. Beyond that, the three critical experiments described in Chapter 8 investigated the effects of rule induction and associative processes on pro- and retro-active interference in the learning process in an attempt to discern whether older people overgeneralised between stimuli and the reasons for this. All experiments used background test results (see Chapter 5) to take account of general factors that may influence cognitive ageing. The AH4 Total was used as an index of fluid intelligence, the Mill Hill Vocabulary Scale (MHV) as a test of crystallised intelligence, the MacQuarrie Test of Mechanical Ability (McQ) measured sensori-motor ability, a Digit Cancellation (DC) task quantified perceptual speed, and Years of Education (YE) was an indicator of socio-economic status and the extent to which participants had used their cognitive abilities through their lifetime. These variables, along with Age itself, were entered into initial correlational and subsequent multiple regression analyses with Overall Accuracy (OA), Final Trial Accuracy (FTA), and in some instances, interference, or change in contingency judgements. This chapter will first summarise the results before going on to examine their relationships with the theories discussed in Chapters 1 to 4 and making some suggestions for further research.

9.1.1: Experiments 1 to 5; Negative and Positive Patterning Problems

Experiment 1 featured ten presentations of two NPPs and two PPPs with four possible allergy outcomes as well as the possibility of a no allergy outcome in response to food stimuli. Here there were overall deficits in speed of learning NPPs relative to PPPs and compound stimuli relative to elemental stimuli. Older participants also acquired the contingencies more slowly, although there were no overall age differences. By the final trial the Young had learned both problems, the Young Old (YO: 55-75) had learned the PPP and the Old Old (OO: 75+) had learned neither problem. By this stage, there were no age differences in learning elements but age related deficits were apparent with respect to compound learning. The multiple regression analysis provided no single predictor of FTA but YE was apparent as a predictor of OA.

Experiment 2 similarly featured ten presentations of two NPPs and two PPPs with the sole manipulation of reducing the number of outcomes to two: allergy or no allergy. This manipulation led to the observation of overall age deficits in learning, particularly between Y and OO groups. Older participants also learned more slowly, especially the NPP, although there were no differences with regard to element and compound learning. By the final trial both Y and YO groups discriminated significantly between the opposing outcomes of elements and compounds but the OO did not. Only a minority of participants in the younger group successfully identified a rule of conjunction to help them predict the outcomes whereas none of the older groups did. Those younger participants who induced the rule scored better on the AH4 Total than the rest of their age cohort and, in addition, better on the DC task than other participants, although the latter observation may be due to age differences between the Rule Correct (RC) and the Rule Incorrect (RI) groups. The RC group learned the contingencies more quickly and were significantly more accurate by the final trial, although not overall. The outcome of the multiple regression analysis was that Age itself predicted both FTA and OA in this experiment.

Experiment 3 sought to give older participants more of a chance to induce rules and consisted of fifteen presentations of one NPP and one PPP. Here the overall age differences disappeared, replaced by an age related deficit in learning the NPP. Indeed the OO group still did not learn the NPP overall, although both other age groups did, and all age groups learned the PPP. There was an overall deficit in learning compounds relative to elements but no significant age differences in this regard. Rule induction was more widespread and there were no significant age differences between RC and RI groups, although only one of the OO group successfully reported the rule. Once again, the RC group scored better on the AH4 Total and DC tasks, although the difference was bigger for the former than the latter. The RC group also learned the contingencies more quickly although there were no interactions between rule group and problem or stimulus. The multiple regression analysis showed that Age itself was a significant predictor of OA but not FTA.

To look at whether one could regard presenting two different problems concurrently as increasing complexity and to ascertain how much one could reasonably expect an ageing population to learn, the two older age groups also participated in Experiments 4 and 5. Both experiments presented stimuli and responses fifteen times and Experiment 4 consisted of two concurrent NPPs whereas Experiment 5 featured two concurrent PPPs. In Experiment 4, by contrast with Experiment 3, both YO and OO groups learned two concurrent NPPs successfully, underlining the relative ease with which two similar problems presented concurrently were learned relative to two different problems, although elements were better learned than compounds. This suggests that presenting different problem types concurrently may be seen as increasing the 'complexity' of a task, relative to presenting two similar problems concurrently, particularly as the memory demands of Experiment 4 were no greater than those demanded by Experiment 3. Experiment 5 derived similar results in that age differences in learning two concurrent PPPs were non-existent although participants learned the contingencies associated with elements more easily than those associated with compound stimuli.

Experiments 1 to 5, overall, showed that NPPs were more difficult to learn than PPPs, contingencies associated with elements were easier to learn than those associated with compounds, and that associative learning ability mediated rule induction. Age related declines were observed in learning NPPs relative to PPPs, in overall learning ability and, perhaps as a consequence, rule induction in some instances. They also suggest there are several factors that may contribute to an age by complexity effect in human conditional learning: number of outcomes, number of stimuli, compound rather than elemental stimuli, whether a linear solution is possible or not, and presenting different problems concurrently. This also suggests that simple forgetting cannot explain all the age related decline in conditional learning performance since if this were the case there would be no age differences in performance between Experiments 3, 4, and 5 as the memory load in these experiments was identical.

9.1.2: Experiments 6 to 8: Conditional and Biconditional Problems

Experiment 6 confronted participants with fifteen presentations of concurrent conditional and biconditional problems. Participants learned the linearly soluble conditional problem more quickly and significantly better than the biconditional problem overall. The young group had acquired both conditional and biconditional contingencies by the final trial, the YO only the conditional discriminations and the OO had learned neither. The sole significant predictor of FTA uncovered by the multiple regression analysis was, once again, Age whereas none of the individual difference measures even correlated with OA.

In order to ascertain whether older people's problems with learning the contingencies were due to the greater processing demands of solving more than one problem concurrently Experiment 7 presented the older groups with a biconditional problem and Experiment 8 a conditional discrimination. There were no overall age effects in Experiment 7 but the OO group still had not mastered the biconditional problem by the final trial. Both YO and OO groups mastered the conditional discrimination in Experiment 8 and there were no age effects.

These experiments more clearly demonstrated that older people are particularly disadvantaged when tackling non-linear problems, although younger participants were similarly compromised by the biconditional relative to the conditional problem. This provides further confirmation that non-linearity can be viewed as increasing the complexity of a problem and that the use of configural generalisation processes may require greater processing resources than elemental strategies. The differences between the problems may, however, be exacerbated by the fact that one only needs to learn contingencies for two elements to acquire the conditional discrimination whereas there are no short cuts to learning the biconditional discrimination.

9.1.3: Experiments 9 and 10; Rule Shift Experiments

In Experiment 9 participants were first given two concurrent PPPs to learn in Stage 1. Following this one of the problems was re-valued in Stage 2 as a NPP and another NPP was presented concurrently using novel stimuli. During the Test Stage participants were given unreinforced exposure to Stage 1 stimuli. Experiment 10 was a direct replication using identical stimuli, the only difference being that the more difficult NPPs were presented during Stage 1 and Stage 2 consisted of PPPs. Following both experiments, participants were asked to complete a compound recognition task consisting of the three compounds they had seen during the experiment and three novel compounds.

In Experiment 9, all age groups learned the two PPPs in Stage 1, although there were overall age related deficits. Age and YE predicted OA whereas FTA was predicted solely by YE. The Stage 2 results were more interesting in that age related deficits were greater for the YO than the OO group and all groups learned the re-valued NPP more slowly than the novel NPP. This suggests that the YO were disadvantaged because they had learned the Stage 1 contingencies better than the OO group and therefore persevered in their incorrect responses longer whereas younger participants' more flexible cognition allowed them to reverse the contingencies more easily. Despite this, by the end of Stage 2 all groups were significantly discriminating between elements and compounds in both problems. For the re-valued NPP, predictors for OA were Age and AH4 Total whereas YE predicted FTA. For the novel NPP AH4 Total predicted OA, but there were no significant predictors of FTA. Test Stage responses were almost entirely unexpected in that younger participants almost exclusively re-valued the Stage 1 contingencies unseen during Stage 2 to match the problem that had been re-valued as a NPP and therefore suffered the greatest retroactive interference whereas the YO suffered least. The differences between Y and YO responses were significant overall. Multiple regression analysis showed that Age, AH4 Total, and YE predicted the extent of change to Stage 1 contingencies not seen during Stage 2. The Young were also very consistent in their responses whereas older participants were not, suggesting quicker extinction of stimulus-response links or greater confusion for older people. There were also age related deficits in compound recognition and false recognition between Y and OO groups.

The results from Experiment 10 were initially similar, although age differences were slightly greater due to the greater difficulty of the NPPs and significant between Y and OO. By the end of the stage, however, all groups were discriminating between elements and compounds successfully. There were no predictors of OA but Age, AH4 Total, and MHV predicted FTA. Stage 2 results were more complex than in the previous experiment in that greater proactive interference effects were apparent, and the young group's responses were more accurate than both older groups, especially for the re-valued problem and again differences were greatest between Y and YO. Despite this all groups' responses to elements and compounds were significantly different by the end of the stage. For Stage 2 OA for the re-valued problem was predicted by AH4 Total and YE whereas OA for the novel PPP was predicted by YE alone, and there were no significant predictors for FTA in either problem. Test Stage responses were profoundly different from the previous experiment despite the minimal manipulation of swapping the order of the first two stages. Younger participants' responses were less consistent than previously and some of them, at least, discriminated between the two problems, unlike in the previous experiment. This suggests that younger participants may have been using a variety of strategies, both configural and rule based, in order to make predictions in the absence of reinforcement. Overall levels of change to the Stage 1 contingencies were less than before and although the Y and YO participants suffered most interference there were no significant age differences in this regard. The extent of change to Stage 1 CD problem contingencies was predicted by the amount of time participants had spent completing Stage 2, although in this instance the young had taken longest so conclusions about the impact of this observation are difficult to make and are unlikely to reflect greater forgetting over time with age. In this experiment there were age related differences in terms of false recognition, but not in compound recognition.

Overall, these experiments have shown that the 55-75 age group may be more prone to proactive interference because they learned the Stage 1 contingencies better than the OO but lacked the cognitive flexibility of the young that allowed the latter to adapt quickly to the re-valued problem in Stage 2. It seemed apparent that older participants were suffering greater proactive interference consistent with elemental generalisation processes than younger participants, perhaps as a result of a failure of inhibitory processes in the former group. There were also greater age differences when problem order reversed from Hard to Easy (NPP to PPP) than from Easy to Hard (PPP to NPP), consistent with observations in both discrimination learning experiments with animals (e.g. Gluck & Myers, 1995, 2001) and humans (e.g. Suret et al. 2003) and in problem solving (e.g. Novick et al. 1991). Test stage responses suggest that when problems shift from Easy to Hard younger participants generalise responses from Stage 2 to the unrevalued stimuli from Stage 1 during test but that this effect does not extend to older people or when problems shift from Hard to Easy. This implies that associative processes can only go so far in explaining how humans respond during conditional learning experiments but that rule induction is contingent on acquisition of contingencies through those associative processes since younger participants learned contingencies better and were more prone to rule based retroactive interference. Fundamentally, this implies that basic associative learning processes underlie rule induction but that heuristic rule use over-rules associative learning in the final analysis.

9.1.4: Associative Interference; Experiment 11

This experiment, again, consisted of three stages two of which presented participants with reinforced compounds and one unreinforced test stage. Stage 1 contingencies were AB+, CD+, EF-, and GH-; Stage 2 contingencies were AC-, BD-, IJ+, and KL+; and Test compounds were AB, CD, EF, GH from Stage 1 and two novel compounds AD and BC. Note that each stage is linearly soluble within itself but that over the first two stages contingencies involving elements A, B, C, and D constituted a biconditional problem. The question here is whether we would observe the same kind of proactive interference between Stage 1 and 2 and whether learning Stage 2 AC-, BD- contingencies would interfere with Stage 1 AB+, CD+ learning more than EF-, GH- and, furthermore, how would participants generalise learnt responses to the novel test compounds AD and BC?

In Stage 1 the young group discriminated significantly more between stimuli associated with an allergy and those associated with no allergy than both the YO and OO groups although all groups' discriminations were significant by the end of the stage. In this part of the experiment, YE predicted both OA and FTA.

During Stage 2 the Young were again significantly more accurate than YO and OO groups. Participants overall were slower to learn the AC-, BD- contingency but older volunteers were particularly compromised in this regard. It is interesting to note that the OO were the slowest to adapt to the revaluation, by contrast to the previous two experiments where it was the YO who experienced the greatest proactive interference. This suggests that there may be a fundamental difference between reversing a rule and reversing learned associations. Despite this, all age groups were successfully discriminating between allergy and no allergy compounds by the end of the stage. For the AC- and BD- stimuli Age and AH4 Total were significant predictors of OA and AH4 Total predicted FTA. For the IJ+, KL+ stimuli, on the other hand, Age itself predicted OA whereas YE predicted FTA. The young group's Stage 2 responses conformed to the Predictions of Pearce's (1994) configural model, whereas older participants' learning suggested the use of elemental strategies.

Test Stage comparisons between AB/CD and EF/GH stimuli showed that older people were less consistent in the way they responded and discriminated less between responses to the two stimulus types. In particular, the OO group did not preserve the discrimination they had learned in Stage 1, suggesting an age related vulnerability to retroactive interference and a greater tendency to use an elemental strategy of summation. Although an age related trend was apparent in the difference between Stage 1 final trial and average Test Stage responses to AB and CD compounds this was not significant and the extent of this interference was weakly predicted by AH4 Total scores. Younger participants preserved the discrimination between AB/CD and EF/GH, as predicted by configural theories.

Analysis of responses to AD/BC stimuli showed there was an age related deficit in the ability to differentiate between these and AB/CD stimuli, although only the YO group failed to discriminate significantly between them. This suggests that younger participants were likely to use a configural learning strategy to extrapolate novel predictions from existing learning, whereas older participants were, again, more likely to use elemental strategies. Analysis of the compound recognition task showed that the younger group recognised significantly more compounds and had significantly greater false recognition than both older groups.

Overall, this final experiment showed again that older people are more vulnerable to pro- and retro-active interference, although in this instance without the influence of rules this disadvantage was monotonic rather than applying more to those who learned initial contingencies better. It also showed that older people were more likely to use an elemental strategy of summation to generalise associative strength from one stimulus to another, whereas younger people tended to use a configural approach and were able to inhibit the extent of perceptual generalisation between stimuli if necessary.

The remainder of this final chapter will be devoted to looking at the implications of these findings for the theories of associative learning and cognitive ageing discussed in the first four chapters. Firstly, consideration will be given to the implication of these data for learning theories. Subsequently general theories of ageing will be looked at, with particular attention being paid to the processing speed (Salthouse, 1996), frontal lobe (West, 1996), and associative deficit (Naveh-Benjamin, 2000) theories. Lastly the question of whether older people overgeneralise between stimuli and use elemental strategies more than younger people and how the results fit in with the theories of learning discussed in Chapters 1 and 2 will be deliberated.

9.2: Implications for Theories of Associative Learning

Whilst the focus of this thesis is on cognitive ageing and to begin to develop an explanation for age related decline in human conditional learning (HCL) ability some of the data have implications for associative learning theory that are worth considering. The results are especially problematic for elemental theories since they reinforce the assertion of Shanks, Charles, Darby and Azmi (1998), and Shanks, Darby and Charles (1998) that younger participants generally use configural strategies to learn HCL discriminations. The strongest evidence for this was Experiment 11, where the test stage responses of young participants were predictable by a slightly modified version of Pearce’s (1994) configural model, but not by elemental models such as Rescorla’s (1973) unique cue theory. Added to this was the replication, in Experiments 1-8, of observations that younger participants were able to solve a number of non-linear problems, although this learning was equally predictable by unique cue theories. More recent elemental theories such as the LePelley (2004) and Wagner (2003) models simply either add factors that may affect the learning rate, such as attention and salience in the former case, or state that stimuli may be encoded configurally if they are from different modalities in the latter case. The present data suggest that elemental models may not be entirely applicable to younger participants' responses during conditional learning experiments. Having said that HCL does, as De Houwer and Beckers (2002) point out, sometimes require elemental assumptions since empirical phenomena such as blocking and summation still occur, these phenomena are, as seen in Chapter 2, equally predictable by Pearce's (1987, 1994, 2002) configural models of learning. There is, therefore, nothing to contradict the assumption that generalisation processes in younger people are configural, rather than elemental.

Older participants, on the other hand, seemed to take a more elemental approach to contingency learning. Recall Experiment 1 demonstrated that the 55-74 age group (YO) could learn PPPs but not NPPs, whereas the over 75s (OO) learned neither. Experiment 2 reduced the number of possible outcomes to two rather than 5 and the YO were able to learn the contingencies, but not the OO. In Experiment 3 the OO could still not learn a NPP, although the YO could. This suggests an increase in the use of simple elemental learning strategies with age, and a commensurate decrease in the ability to use more sophisticated configural learning approaches. It could, however, be argued that Experiment 4 demonstrated that even the OO could learn two concurrent NPPs, but this can be solved by the simple application of a rule that states elements lead to an outcome, whereas compounds do not. This is clearly a less sophisticated rule than the rule of conjunction necessary for solution when a conflicting PPP is present and may be within the ability of the oldest participants. This view is underlined by the observation that since the OO could not solve a biconditional problem when presented alone in Experiment 7. On the face of it the relatively low number of exemplars present in Experiment 7 should make it easier than Experiment 4, but the difficulty is in the absence of a clear rule or strategy that may be less demanding of cognitive resources than configural or unique cue approaches that makes this problem so difficult. Certainly when this problem was combined with a conditional problem during Experiment 6 a similar pattern of observations to Experiment 1 emerged in that younger participants learned both conditional and biconditional problems, the YO learned the linearly soluble conditional but not the biconditional problem, and the OO learned neither. This, again, suggests a progressive, age-related deterioration in the ability to use configural processing in solving conditional learning problems.

The real problems for the validity of applying associative learning theories to HCL come, however, from the rule reversal effects in Experiments 9 and 10. In the former experiment, those participants best at learning the problems generalised the Stage 2 responses to stimuli unseen since Stage 1 at test. This observation is wholly inexplicable by any form of associative learning theory since both configural and elemental theories do not predict a change in contingencies if the same or perceptually similar stimuli are not present. Participants' responses at test may, therefore, have more in common with problem solving. Despite this, there is some support for Shanks and Darby's (1998) notion that people who acquire contingencies more quickly and accurately are more likely to induce rules (see Experiments 2 and 3). This, in conjunction with the observation that rule use was the best explanation of younger participants' test responses in Experiments 9 and 10, suggests that associative learning theory may be adequate to describe initial learning but that once participants start, rightly or wrongly, to induce generalisable rules this overshadows any associative learning that may have taken place. It may be that there is some correspondence between HCL and problem solving since both avenues of enquiry have demonstrated that learning an easy problem facilitates the solution of a harder problem (HCL: Suret et al. 2003; Problem Solving: Weisberg & Alba, 1981; Novick & Holyoak, 1991) and that generalisable, rule based solutions are dependent on background learning (Shanks & Darby, 1998; Novick & Sherman, 2003). Furthermore both seem to have the same fundamental neural bases in the hippocampal and frontal regions of the brain (see Chapter 2). Of course, this correspondence is entirely speculative at present and would need much more research for any firm conclusions to be drawn but it may be an interesting and fruitful avenue of investigation. These questions are, however, not the central concern of the present research and we will now move on to what are the fundamental questions to be addressed with regard to cognitive ageing.

9.3: Implications for Theories of Cognitive Ageing

The research presented here attempted to address a number of questions. Firstly, and most simply, whether HCL is an age sensitive cognitive ability. Beyond this, one could ask whether HCL ability is dissociable from other cognitive and sensori-motor abilities and if participants' responses conform to the patterns expected by Processing Speed theory (Salthouse, 1996), Frontal Lobe theory (West, 1996), or the Associative Deficit hypothesis (Naveh-Benjamin, 2000).

9.3.1: Is HCL Age Sensitive?

From the results of the preceding experiments, it is probably safe to conclude that HCL ability is age sensitive and shows a decline with age. The only experiments that showed no evidence of age related decline were Experiment 5, which featured two concurrent PPPs, and Experiment 8, which consisted of a single conditional problem. Note that in both these experiments only YO and OO groups participated, and that age differences were apparent when participants completed two concurrent PPPs in Stage 1 of Experiment 9. One could also note that the only problems that did not suggest age related differences were linearly, and therefore elementally, soluble. Again, this implies that older participants were using an elemental approach to generalisation.

9.3.2: Is HCL Ability Dissociable From Other Age Sensitive Abilities?

One of the biggest questions in cognitive ageing is whether all age sensitive abilities are subsumed by factors that are more general, such as fluid intelligence or processing speed, or whether they are independent of one another. The current research addressed this question by collecting background data (see Chapter 5) on a number of measures designed to reflect general factors that have been suggested to underlie all or much of general age related cognitive decline. Variables included in these analyses were Age, AH4 Total (AH4), Years of Education (YE), Mill Hill Vocabulary Scale (MHV), MacQuarrie Test of Mechanical Ability (McQ), Digit Cancellation (DC), and Stage 2 Time for Experiments 9, 10, and 11. These measures were then entered into multiple regression analyses if they correlated with Overall Accuracy (OA), Final Trial Accuracy (FTA), or estimates of interference. The results of these multiple regressions will now be used to assess the extent to which age itself or other factors contribute to HCL. Table 9.1 summarises the findings of this part of the investigation. These observations may also help to address some of the theories mentioned in Chapters 3 and 4. These include fluid intelligence explanations, disuse theories, sensori-motor deficits, and processing speed theory.

Table 9.1: Predictor Variables Identified by Multiple Regression for Overall Accuracy (OA), Final Trial Accuracy (FTA), and Interference

Experiment		OA	FTA	Interference
1		YE		
2		Age	Age	
3		Age		
6			Age	
9 Stage 1		Age, YE	YE	
9 Stage 2	AB	Age, AH4	YE	
	EF	AH4		
9 Test				Age, YE
10 Stage 1			Age, AH4, MHV	
10 Stage 2	AB	AH4, YE		
	EF	YE		
10 Test				Stage 2 Time
11 Stage 1		YE	YE	
11 Stage 2	AB/CD	Age, AH4	AH4	
	IJ/KL	Age	YE	
11 Test				AH4

9.3.2.1: Age

There was some evidence to suggest that age itself was a predictor of response accuracy in a number of experiments and that HCL may therefore be dissociable from other abilities that exhibit an age related decline. Age was a significant individual negative predictor of OA in Experiments 2, 3, 9 (Stage 1 and AB problem Stage 2) and 11 (Stage 2), and of FTA in Experiments 2, 6, and 10 (Stage 1). In this sense age seems to predict accuracy more within the concurrent negative and positive patterning problems of Experiments 2 and 3 and in the concurrent biconditional and conditional problems of Experiment 6 since in all other instances other age related variables were also predictors. This indicates that the ability to complete two different problems simultaneously may be particularly, and perhaps uniquely, sensitive to the ageing process. This suggests that solving concurrent but different problems constitutes complexity, and the observed age related difficulties may well be related to a decline in underlying cognitive resources. Age also positively predicted the extent of change to the CD problem between the end of Stage 1 and test in Experiment 9; although this is almost certainly due to the fact that younger participants' responses suffered the greatest retroactive interference through rule based generalisation rather than being related to cognitive ageing as such. Not only this, but age differences and interactions in some experiments were preserved when AH4 scores were entered as a covariate (Experiment 1, Age by Blocks; Experiment 2, Age, Age by Blocks; Experiment 3, Age by Problem; Experiment 9, Test stage, CD Change, Hits; Experiment 10, Stage 2 accuracy, Age by Problem by Blocks, Test Stage Age by Problem; Experiment 11 Stage 2 Age by Problem by Blocks, Test Stage ABCD vs EFGH Age by Trials, ABCD vs ADBC Age by Stimulus). Again, this suggests that some parts of the observed age differences in conditional learning ability may be independent of general cognitive ability and consequently run contrary to the 'dull hypothesis'. One explanation may be that age differences independent of general intelligence reflects very basic learning processes that do not require higher order processing. Certainly, given that the associative learning explanation of conditional learning relies on an analogy with animal learning it makes sense to suppose that the processes involved in feature, although not necessarily rule, based generalisation may be very basic indeed. There is, however, an alternative possibility: perhaps the problems were simply too abstract for older people in that they relied on the ability to imagine the hypothetical consequences of the presence of imagined foods or words on an abstract reality. Whilst this may sound like the kind of explanation that would be subsumed within a fluid intelligence explanation this may not necessarily be the case. Stuart-Hamilton (1994, pps. 59-61) raises the possibility that older people may be worse at Piagetian tasks than younger people, implying that abstract thinking abilities may decline with age. Again, it would be easy to dismiss this as merely reflecting a change in general intellectual ability, but two more recent studies suggest that some abilities and orientations related to the solution of Piagetian tasks are independent of general intelligence. Firstly, Stuart-Hamilton and McDonald (2001) gave participants a set of Piagetian concrete operational tasks and found that these abilities, rather than fluid or crystallised intelligence or age itself, predicted a measure of need for cognition in a sample of older adults. Whilst it is almost certainly true that need for cognition is probably higher in those who volunteer to take part in cognitive ageing studies this suggests that what individual differences there are in this respect are mediated by cognitive skills unrelated to more general intellectual ability. In a similar vein McDonald and Stuart-Hamilton (2003) found that older participants' tendency to make egocentric responses in a replication of Piaget and Inhelder's (1956) 'Three Mountains Task' was predicted by age, rather than fluid or crytallised intelligence or personality. This suggests that there are some aspects of abstract thinking and problem solving that are independent of mainstream explanations of cognitive ageing. It is certainly possible that older people's poor performance may be undermined by a lack of abstract thinking abilities in several ways: firstly just by virtue of an age related decline in such abilities, and secondly by a decline in motivation toward complex problem solving, or need for cognition. Given the lack of research in this area there is certainly a case for looking at some age related decline afresh with a view to confirming or denying the influence of abstract thought abilities and its relationship with task motivation on the results of cognitive ageing studies.

9.3.2.2: Crystallised and Fluid Intelligence

Fluid Intelligence, or the ability to think flexibly about novel problems, was evaluated with the AH4 Group Test of General Intelligence (AH4: Heim, 1968) whereas Crystallised Intelligence, or knowledge, was assessed with the Mill Hill Vocabulary Scale (MHV: Raven, 1982). This reflects the distinction between fluid and crystallised intelligence made by Cattell (1963; Horn & Cattell, 1967; Horn, 1989; Horn et al. 2000) in that the former decreases with age whereas the latter is either stable or increases with age and both are well standardised tests established as appropriate measures of the two constructs (c.f. Rabbitt, 1993; Rabbitt et al. 2004).

MHV scores proved, unsurprisingly, poor overall predictors of accuracy. The observation that MHV was a negative predictor of FTA in Experiment 10, Stage 1 is explicable when one considers that Age, too, bore a similar relationship with FTA and that MHV scores are also age related. It is, therefore, possible that in this instance age itself was the mediating factor rather than MHV scores *per se*.

AH4 Total scores did not, however, feature as predictors in the single stage experiments, although their inclusion as covariates did diminish the relationship of age to some aspects of learning. In particular more demanding learning involving compounds and nonlinear problems produced results that suggested age related deficits in these areas were due to declines in general cognitive abilities. More interestingly, AH4 scores predicted Stage 2 overall accuracies for re-valued stimuli in Experiments 9, 10, and 11 (albeit in conjunction with Age, YE, and Age respectively), and FTA for the AB/CD stimuli in Stage 2 of Experiment 11 as well as the extent of interference in Experiment 11's Test Stage and FTA in Experiment 10, Stage 1 (concurrent NPPs). It would make sense to suppose that this reflects participants' vulnerability to proactive interference and their use of elemental strategies since these effects are largely confined to re-valued stimuli in Stage 2 of all multiple stage experiments. This interpretation is supported when AH4 was added to analyses as a covariate: this factor accounted for age differences in Stage 2 of Experiment 9, where learning shifted to a more difficult NPP but was less influential in Experiment 10, where learning shifted to the easier and linearly soluble PPP. Importantly, though, it also predicted the extent of retroactive interference at test in Experiment 11 and when added as a covariate attenuated age related differences in resistance to pro and retro active interference. This is understandable on consideration of what the AH4 actually assesses. The test itself really involves the detection of patterns and regularities in numbers, words, and shapes through mental manipulation, series completion, and analogical reasoning. It may be, therefore, that an ability to detect and act on environmental regularities may underlie older participants' relative difficulties with any change in contingencies and tendency to proactive interference in particular, but also to retroactive interference in Experiment 11. This indicates that fluid intelligence as a concept has some credibility, although this cannot explain why fluid intelligence itself should decline with age. Perhaps this relates to frontally mediated perseverative errors in older people, as, for instance, in the Wisconsin

Card Sorting Test (e.g. Dempster et al. 1999) or focus switching in working memory tasks (Verhaeghen et al. 2002, 2005), and would therefore support West's (1996) Frontal Lobe theory of cognitive ageing. It may also suggest that fluid intelligence abilities may underlie the inhibition of perceptual generalisation in order to prevent interference to previously learnt contingencies. The observations that age differences in false recognition were removed by inclusion of AH4 as a covariate also suggests that general intelligence can explain a large proportion of learning deficits. Perhaps the 'dull hypothesis' may not be quite as dull as has been suggested.

9.3.2.3: Disuse Theories

Recall from Chapter 5 that everyday activities were excluded from the experimental analyses since the Everyday Activities Questionnaire (Pushkar et al. 1997) scores bore no relation to background cognitive measures. In the absence of these measures Years of Education may be the best reflection of disuse in the present analysis. This factor is assumed to reflect lifelong cognitive activity since better-educated people tend to be employed in more cognitively demanding jobs. Note, however, that although this makes sense this conjunction of education and career is also associated with higher socio-economic status which may also be a factor militating against age related cognitive decline (Antonucci, 2001; but see Kliegal et al. 2004). Years of Education were predictive of OA in Experiment 1, Stage 1 of Experiments 9 and 11, and for both AB and EF problems in Experiment 10, Stage 2. It also predicted FTA in Experiment 9 Stage 1 and for the AB problem in Stage 2, as well as for Experiment 11 Stage 1 and the IJ and KL stimuli in Stage 2 and, furthermore, predicted the extent of change to Stage 1 CD discriminations in the Test Stage of

Experiment 9. As with AH4 scores this pattern of results suggests that YE is a better predictor in multiple stage experiments, and there is always the possibility that since intelligence tests such as the AH4 were designed to predict academic performance that YE and AH4 attainment may be related. There are, however, real differences in the pattern of results since YE was a predictor of Stage 1 OA and FTA in Experiments 9 and 11, which were both linearly soluble, and in OA for Experiment 1. By way of contrast AH4 scores were more predictive of accuracy when stimuli had been re-valued and may therefore bear a greater relationship to executive abilities than YE. Even when YE predicted Stage 2 accuracy in Experiment 10 this was not confined to the re-valued stimuli, and indeed in Experiment 11, Stage 2, YE predicted accuracy for responses to the novel IJ and KL stimuli. Conclusions are therefore difficult to draw. One could say that YE predicted participants' accuracy in relatively simple problems since it was a significant predictor of accuracy in Stage 1 of Experiments 9 and 11 but this could not explain why it predicted OA in Experiment 1. One explanation could be that younger participants had, on average, more years of education than the older groups and the observed differences reflect this. This makes sense but age itself should be, logically, a co-predictor if this was the case but only OA for Experiment 9, Stage 1 shows age and YE to predict the same thing. Clearly YE seems to be related to response accuracy in some way but it is difficult to see exactly how from the data available and more research needs to be done to illuminate this relationship.

9.3.2.4: Sensori-Motor Deficits

Sensori-motor abilities were assessed with the MacQuarrie Test of Mechanical Ability (see Chapter 5). There was no evidence that this factor predicted learning ability. It can be concluded, therefore, that sensori-motor deficits have little to do with HCL ability in the present context.

9.3.2.5: Processing Speed Theory

There is almost no evidence that performance on the DC task predicted learning ability at all. DC performance significantly correlated with OA or FTA in a number of experiments, and constituted part of significant multiple regression models on those occasions. Despite that, DC never emerged as an individually significant predictor of learning accuracy or interference. This suggests that perceptual speed had little to do with HCL ability. Having said that one must bear in mind that the current research relied on a single measure of processing speed, whereas Salthouse (1996; 2000) and other researchers tend to use batteries of tests designed to operationalise the construct, although exactly what ‘processing speed’ is and exactly what these test batteries measure is open to question (see Parkin & Java, 1999, 2000). One cannot completely rule out the processing speed theory merely because DC cannot predict HCL, but the present evidence suggests that it cannot explain the current data.

So far, a number of theories have been assessed using the background measures obtained from participants that were detailed in Chapter 5. These included whether HCL ability was age sensitive and whether it was dissociable from other cognitive abilities. Furthermore, individual difference measures were combined with experimental data to assess the extent to which HCL could be predicted by other abilities that commonly decline with age. This included the relative contribution of fluid and crystallised intelligence, disuse theories, sensori-motor deficits, and processing speed. What it neglected was the relative contributions of frontal lobe theory and the associative deficit hypothesis. These were not assessed directly since the main aim of the research was to establish a body of data on age related deficits in HCL in the absence of any other. The contribution of frontal and medial temporal lobe areas of the brain to learning and memory (see Chapter 2) and the nature of their age related decline (see Chapter 4) is well documented, so it should be possible to assess the theories by examining the pattern of age related deficits apparent in the experiments. The next section, therefore, will examine both of these perspectives in relation to experimental data.

9.4.1: The Frontal Lobe Theory of Cognitive Ageing

Recall from Chapters 2 and 4 that frontal areas of the brain seem particularly prone to the effects of ageing (e.g. Woodruff-Pak, 1997; Raz, 2000; Shan et al. 2005). Frontal lobe (FL) areas are generally associated with executive control of cognitive processes, planning, decision-making, attention, and using experience to guide future behaviour (e.g. Shallice & Burgess, 1991; Bechara et al. 2000). Other cognitive deficits associated with FL damage include a vulnerability to perseverative errors (e.g. Gunning-Dixon et al. 2003), differentiating between stimuli with different outcomes and task relevant and task irrelevant stimuli (e.g. Dimitrov et al. 1999), effortful encoding, and retrieval processes (e.g. Tulving, 2002), reversal learning (e.g. Kringelbach et al. 2003), learning about the reward value of stimuli (e.g. Rolls, 2000; 2004), and inhibiting prepotent responses (e.g. Damasio, 1994; Milham et al. 2002). Recall, from Chapter 4, that this pattern of deficits is apparent in older people and that West's (1996) FL theory of cognitive ageing suggests that it is these deficits that underlie most, if not all, of the observed age related cognitive decline. Remember, too, from Chapter 2 that the work of Rolls (2000, 2004; Francis et al.

1999) suggests that FL areas are vital for the formation of stimulus-response links and the conscious differentiation of stimuli with different outcomes. Predictions that may be derived from this theory relevant to the FL theory of cognitive ageing are that older participants should exhibit deficits in stimulus response learning and that they should overgeneralise between stimuli because they lack the cognitive resources necessary to initiate configural learning processes that appear necessary for the solution of non-linear problems and resistance to feature based interference. Furthermore, because older participants learn more slowly they should have difficulty inducing rules (cf. Shanks & Darby, 1998) and should recover more slowly from the proactive interference resulting from contingency reversals, persevering with previously valid contingencies longer than younger participants. One should also see age deficits increase as processing load or complexity increases since more effortful forms of processing appear to be more affected by increasing age and FL deficits.

There is little to contradict these predictions in the research reported here. Certainly, in all but the least complex of the experiments (Experiments 4, 5, and 8) there were observed age deficits in contingency learning. This suggests that only when cognitive effort was increased did the age related deficits become apparent. Again, this is consistent with FL theory but also with the 'age by complexity' effect (see Chapter 3), although it is apparent that the two may be related or even synonymous given the FL deficits associated with effortful processing (e.g. Tulving, 2002).

Furthermore, as complexity increased, as defined by the number of different problems or outcomes or linear insolubility of the problems, so did age differences. There is little doubt that older participants overgeneralised between stimuli because of their poor performance in biconditional relative to conditional problems and NPPs relative to PPPs. This implies that non-linear problems may engage more processing resources and cognitive effort than linear problems and those deficits may therefore be because of FL atrophy. An alternative, and less effortful strategy to adopt may be to apply a rule of summation that would render non-linear problems insoluble but would be consistent with older people's reliance on stereotypical, schematic responses that reduce processing load (Mather et al. 2003). This use of summation as a strategy resulting in overgeneralisation is further evidenced by test stage responses in Experiment 11: older participants tended to be more subject to retroactive interference and confused about which stimuli led to which responses. Younger participants, on the other hand, made responses more consistent with configural generalisation processes. Older people also differentiated less between responses to stimuli seen in Stage 1 (AB, CD) and novel stimuli made up of the same elements (AD, BC). Although younger participants seemed to overgeneralise more in Experiment 9 this was almost certainly due to their having learned an heuristic during Stage 2 that was applied to all test stimuli, and it is arguable that simply inducing and applying a generalisable rule may utilise effortful processing resources.

It was also certainly the case that older participants recovered from proactive interference more slowly than younger people did. This was consistently observed through the three critical experiments (9, 10, and 11), although this effect was complicated because in Experiments 9 and 10 the YO group suffered the greatest proactive interference. Presumably, this was because they had learned the Stage 1 contingencies more than the OO and may therefore have been more prone to induce a partial rule that applied to that stage only and consequently more likely to carry on applying that knowledge, whether gained through associative learning or rule induction, because of a predisposition toward FL mediated perseverative errors. This is likely because rule induction itself seems to be age sensitive, if only because of older participants' poorer learning. Not only that, but AH4 Total scores predicted learning accuracy consistently for Stage 2 re-valued problems, suggesting that more fluid, adaptive, reflective processes were required for reversal learning than any other form of learning.

Another point to consider is that Age as a factor was also a good predictor of accuracy in many of the experiments. It is possible that the capacity of age to predict learning may be subsumed by neuropsychological factors not accounted for in the present research, which has concentrated on collecting experimental data and in discounting other more established theories, such as Salthouse's processing speed theory, or the influence of sensori-motor abilities. From the results of the present investigations, however, it is apparent that more research involving extensive neuropsychological testing or in vivo scanning is justified.

It would also be useful to look in more depth at specific process deficits that may underlie older people's tendency to overgeneralise between stimuli. As discussed previously it is possible to explain overgeneralisation in terms of inhibitory failure (c.f. Hasher and Zacks, 1988; Milham et al. 2002) since the preservation of learned discriminations surely requires the inhibition of subsequently learned responses, and avoiding proactive interference almost certainly means suppressing learnt responses to previously seen and perceptually similar stimuli. This explanation gains credence since the assumption that younger participants can inhibit the extent of generalisation of associative strength from perceptually similar stimuli led to accurate predictions for Pearce's (1994) configural model in terms of younger participants' responses in Experiments 6 and 11.

There is, however an equally plausible explanation for these observations in terms of source monitoring (e.g. Johnson et al. 1993; Hedden and Park, 2003; Simons et al. 2004). It is possible that predicting accurate responses to stimuli relies on memory for the context in which contingencies were originally learnt. For instance, in Experiment 11 it may be that younger people preserved the discrimination between AB, CD and EF, GH stimuli at test since they recognised the context in which they were presented as being the same as Stage 1 of the experiment whereas older participants did not. It would, therefore, be necessary to look at the relative contributions of inhibitory and source monitoring processes on resisting interference in contingency learning. This is particularly the case since although the data cannot rule out FL theory and gives us reason to conclude that this is very probably a factor there is no direct evidence for it. Furthermore stimulus-stimulus associations may be adversely affected by age and it is difficult to partial out this possibility at present. The next section, therefore, will look at the evidence for the associative deficit hypothesis.

9.4.2: The Associative Deficit Hypothesis

Recall, from Chapter 2, that the medial temporal lobe (MTL) and in particular the hippocampal region has been implicated in creating integrated multimodal episodic memories that are ultimately stored in cortical areas (e.g. Scoville & Milner, 1957; Marr, 1970, 1971; Squire et al. 1985; Zola-Morgan et al. 1986). In terms of associative learning the formation of a conditioned response is unaffected by hippocampal region damage (e.g. Gabrielli et al. 1995), as is simple category learning (Knowlton et al. 1994). Theorists such as Gluck and Myers (1993; 2001) and Rudy and Sutherland (1995), however, suggest that this region serves the purpose of creating internal representations of complex, or compound, stimuli that can be differentiated from the elements that compose them, and there is much to suggest this is generally accurate (see Chapter 2). At the very least problems with this region should result in mnemonic deficits for compounds or context, or possibly result in learning that conformed to the predictions of elemental theories such as the Rescorla-Wagner (1972) model since simple stimulus-response learning may not be impaired. There is evidence that the MTL and hippocampal region is subject to age related decline (See Chapter 4, e.g. Raz, 2000; Woodruff-Pak, 1997), as are acetylcholine levels in the region (e.g. Terry et al. 2003) that may mediate storage and recall functions (Gluck et al. 1996). The suggestion that learning about stimulus-stimulus associations is contingent on medial temporal lobe areas is consistent with Naveh-Benjamin's (2000; Naveh-Benjamin et al. 2002 Naveh-Benjamin et al. 2003, Naveh-Benjamin et al. 2004) associative deficit hypothesis of cognitive ageing. This suggests that the age related decline in MTL mediated associative processes may explain age related deficits in memory. This hypothesis was tested in two areas of the preceding experiments: compound versus element learning and compound recognition tasks.

There were significant effects of Stimulus in Experiments 1, 3, 4, and 5 which showed that, for all age groups, contingencies associated with elements were easier to learn than those associated with compounds. There were, however, no interactions between Age and Stimulus, suggesting that older people's compound learning was not differentially affected by the ageing process relative to younger people's compound learning deficits. On the other hand, there was the observation in Experiment 1 of significant final trial differences between Y and OO groups in terms of compound, but not element, learning. This observation, however, was not replicated in the other experiments. Overall, therefore, this is at odds with an associative deficit hypothesis that predicts a greater decrement for learning compounds than elements for older people relative to the difference between younger people's element and compound learning. There is, however, an alternative explanation for why the expected differences were not observed. Recall that, according to Gluck and Myers (1993; 2001) the process of predictive differentiation involves creating representations of compounds discrete from the elements that compose them only if feedback from cortical areas indicates that they lead to different outcomes. In this context one could argue that FL deficits in acquiring stimulus-response contingencies could be obscuring any potential associative deficits. It is however, equally, if not more plausible to suggest that learning about compounds *per se* may involve higher-level processes such as inhibition or source monitoring when their outcomes conflict with the outcomes of the elements of which they are composed or with the outcomes of similar compounds, as they did in the experiments reported here. Therefore the observation that element learning is easier than compound learning does not necessarily imply a MTL deficit, and Gluck's model, as observed in Chapter 2, may be overestimating the role of MTL regions and underestimating the role of FL regions in learning.

In terms of compound recognition, on the other hand, the evidence suggests that age related associative deficits might be apparent. In both Experiments 9 and 11 older participants recognised fewer of the test compounds than younger participants did. In Experiments 10 and 11 older participants suffered more false recognition in that they incorrectly identified significantly more compounds as having been seen, after controlling for compound memory, during the experiment when they had not appeared. This provides limited support for the associative deficit hypothesis in the context of HCL, although it is equally clear that this theory alone could not provide a convincing explanation for the pattern of age related differences in HCL ability, particularly since LaVoie et al. (2006) found that FL deficits were associated with false recognition. It seems likely that, as Band et al. (2002) suggested, cognitive decline in general, even in relatively simple contingency learning, is a consequence of physical decline in more than one brain region. The question is of relative contribution, and the present research suggests that the processes underlying HCL are more likely to depend on FL regions, although MTL regions may contribute to compound recognition. As a consequence there is, again, every justification to proceed with full scale neuropsychological testing or in vivo scanning studies in order to gain a better understanding of the relative contributions of FL and MTL areas to human conditional learning.

9.5: Summary of Conclusions

So, what has been learned from the present research about the way humans tackle contingency learning problems and the effect of ageing on this ability? In order to address this question it would be expedient to split it into two parts. Firstly, what are the implications for the applicability of theories of associative learning to Human Conditional Learning? Secondly, what are the consequences of ageing on HCL ability and how do the patterns of decline apparent in the present experiments fit in with existing theory?

In answer to the first question it is apparent that associative learning theories can predict some, but not all, HCL ability. In common with animal learning theories, there are some empirical observations best explained using elemental theories, and some best explained using configural theories. Both elemental and configural approaches have already been tested by Shanks, Charles, Darby, and Azmi (1998) and Shanks, Darby, and Charles (1998) and been found unable to predict the extent of resistance to retroactive interference in HCL under some circumstances, although configural theories best predicted responses in Shanks et al.'s studies.

The present research does nothing to disconfirm those conclusions, the test stage results of Experiments 9, and 10 being inexplicable by elemental or configural theories since participants seemed to have been relying on rule, rather than feature based generalisation. The observations of rule based, rather than feature based, generalisation reported in Shanks and Darby (1998), however, seem to have been largely ignored in the associative learning literature, perhaps because of the profound difficulties they raise for the application of associative learning theories to HCL. Recall that they suggest that rule induction overshadows associative learning when it occurs, although rule induction itself is contingent on what has been learned about stimulus response contingencies in that faster, more accurate learning seems to predict rule induction. The present series of studies merely confirms this suspicion, since rule correct groups in all experiments, where it was tested, learned contingencies more quickly and accurately than rule incorrect groups.

Furthermore, Experiments 9 and 10 demonstrate that problem order has a profound effect on HCL judgements to an extent entirely unpredictable by associative learning theories and, indeed, it may be that once rule induction has occurred responses may be better predicted by appealing to the problem solving literature. Despite this, however, it is clear that the initial acquisition of contingencies may still be best predicted by associative learning theories. The predictions of Pearce's (1987, 1994, 2002) configural learning theory accurately predicted younger participants' responses in experiments that did not offer the opportunity of rule-based generalisation, especially Experiments 6 and 11. One could, therefore, conclude that associative learning theory still has a place in explaining HCL ability but researchers should be aware that this approach may be limited if the possibility of rule induction and analogical transfer of solutions exists.

In terms of the second set of issues around cognitive ageing that constituted the major reason for the present research it seems that a paper and pencil version of the food-allergy paradigm is a valid methodology for this area of investigation since participants of all ages could acquire stimulus-response contingencies. It was equally apparent that the task is sensitive to cognitive ageing in that an age related deficit in learning ability was evident, and the results suggest a number of explanations for this decline and appear to disconfirm some potential explanations as well.

Firstly, the seemingly somewhat archaic fluid intelligence explanation seemed to predict some HCL abilities well through the observation that AH4 scores predicted accuracy in some circumstances. This observation was particularly consistent in reversal learning in Experiments 9, 10, and 11, where Stage 1 contingencies were re-valued in Stage 2. Given that FL theory would predict a greater vulnerability to proactive interference due to difficulties with reversal learning due to perseverative errors and a compromised ability to inhibit previously learned responses it may be that the abilities tested in the AH4 might be mediated by FL declines. Equally AH4 scores alone predicted resistance to retroactive interference in Experiment 11, again this eventuality would be predicted by FL theories in terms of either inhibitory or source monitoring deficits, and one might suppose that there was a relationship between what is termed frontal lobe or executive function and fluid intelligence.

Although this suggestion may seem purely speculative, it is not without foundation. Duncan (2005; 1995; Duncan, Burgess, & Emslie, 1995) has found that measures of fluid intelligence such as Cattell and Cattell's (1960) culture fair intelligence test are able to predict frontal lobe functions as accurately as neuropsychological tests. In this view, the terms 'executive' or 'frontal' functions seem synonymous with fluid intelligence and the observations made above would indeed suggest that resistance to pro- and retro-active interference in HCL is mediated by executive or frontal abilities.

On the other hand, other researchers are more cautious in interpreting these kinds of observations. Rabbitt, Lowe, and Shilling (2001), for instance (see also Rabbitt & Lowe, 2000) argue that correlations between scores on the Culture Fair Intelligence Test and neuropsychological tests of executive function reflect the fact that fluid intelligence tests do indeed reflect a general intellectual ability and that therefore these correlations should not be surprising. In this conception, therefore, executive abilities are part of a subset of abilities subsumed by general fluid intelligence. Note, however, that Rabbitt (2005) has softened his stance in the light of fMRI evidence from Duncan and Owen (2000) suggesting that frontal areas were preferentially activated by fluid intelligence tasks. If this is, indeed, the case then the observation that pro- and retro-active interference are mediated by fluid intelligence constitutes reasonably strong evidence that this age sensitive ability is contingent on the integrity of the frontal lobes.

There was, in addition, limited evidence from the compound recognition tasks that memory for compounds was subject to an age related decline, giving some restricted support to an associative deficit hypothesis.

9.6: Directions for Further Research

As with the vast majority of research, the present series of experiments raises more questions than it answers. Although the present research focussed on cognitive ageing the observations made with regard to the effects of rule induction and the parallels between problem solving and HCL in connection with Easy-Hard transfer should not be ignored. Certainly, there has been no effort to integrate the findings of Shanks and Darby (1998) into associative learning theories since their publication and the present research reinforces their findings. There is, therefore, a clear gap to be filled in determining the extent to which associative processes underlie causal learning. Beyond this the elemental versus configural debate rumbles on in the literature (see Rescorla, 2003, Wagner, 2003, and LePelley, 2004 for recent examples), and it has been seen that younger adults' performance in HCL tasks may be best predicted by configural models of learning whereas older people's responses are more elemental in nature. This, naturally, leads to the question of generalisation and suggests researchers' efforts might be usefully employed in the task of establishing precisely how older adults generalise between stimuli and explore age differences in this regard. Some of this work has already been carried out in the domains of time perception (e.g. Wearden et al. 1997; McCormack et al. 1999) and conceptual generalisation (e.g. LaVoie et al. 2006), although not all modalities or stimulus types may be subject to increasing overgeneralisation during later life so there is a justification for extending this work and exploring potential fundamental cognitive abilities that may underlie and predict generalisation processes.

In terms of cognitive ageing there is a clear justification for carrying out further research into HCL ability as it is clearly an area that is subject to cognitive decline in old age. There is evidence that implies that HCL ability may be related to FL and MTL mediated abilities and there is therefore a need to carry out more research that includes comprehensive batteries of neuropsychological tests. This would enable us to assess the relative contribution of these areas to associative learning in general and in predicting the extent of age related decline in particular. Of course, it would clearly be preferable to take an in vivo scanning approach but this would be contingent on access to these kinds of facilities. The collection of more comprehensive background data on participants would also enable us to gauge more accurately the extent to which HCL ability in general, and generalisation in particular, is dissociable from other age vulnerable cognitive abilities.

One could also gather more evidence that older adults tend to use elemental strategies to learn contingencies and generalise from them, rather than configural strategies younger participants seem to favour. Since this implies that configural learning requires more processing resources this opens up another avenue of enquiry.

In addition, one could address the questions of whether processing speed is unrelated to HCL ability, and what exactly it is about years of education and AH4 ability that makes them able to predict different aspects of the learning tasks. In the latter case, especially, there is a real question mark over whether fluid intelligence is, in fact, dissociable from frontal or executive abilities. Future research could therefore analyse the relationships between fluid intelligence, neuropsychological test scores, and HCL ability in order to reconcile these observations. Given that there seem to be aspects of conditional learning that are independent of fluid intelligence it would also be necessary to explore these areas. It may be, as discussed earlier, that these unexplained aspects are fundamental associative learning processes or they may be better explained by a decrement in abstract thinking skills that are independent of fluid intelligence, as demonstrated in studies of age related decline in Piagetian tasks (e.g. McDonald & Stuart-Hamilton, 2003).Overall, the present research has successfully opened up another area of enquiry to those interested in cognitive ageing and has suggested ways in which the current findings can be incorporated into future research.

10.1: References

Albert, M.S., Wolfe, J., & LaFleche, G. (1990). Differences in abstraction ability with age. *Psychology of Aging*, *5(1)*, 94-100.

Andres, P., Van der Linden, M., & Parmentier, F.B.R. (2004). Directed forgetting in working memory: age-related differences. *Memory*, *12*, 248-256.

Antonucci, T.C. (2001). Social relations: An examination of social networks, social support, and sense of control. In Birren, J.E., Schaie, K.W. (Eds.), *Handbook of the Psychology of Aging, 5th Edition* (pp. 427-454). San Diego, CA: Academic Press.

Ardila, A., Ostrosky-Solis, F., Rosselli, M., & Gómez, C. (2001). Age-related cognitive decline during normal aging: the complex effect of education. *Archives of Clinical Neuropsychology*, *15(6)*, 495-513.

Aristotle (350 BC). *On Memory and Reminiscence*. (J.I. Beare, Trans.). (Reprinted from Ross W.D. (Ed) (1931). *The Works of Aristotle, Vol. III*, Chapter 2, Oxford: Clarendon Press.) In R.J. Herrnstein & E.G. Boring (Eds.), (1965). *A Source Book in the History of Psychology* (pp. 327-330). Cambridge, MA: Harvard University Press.

Atkinson, R.C., & Estes, W.K. (1963). Stimulus Sampling Theory. In Luce, R.D., Bush, R.R., & Galenter, E. (Eds.), *Handbook of Mathematical Psychology, Volume 2* (pp.121-268). New York: Wiley.

Atkinson, R.C., & Shiffrin, R.M. (1968). Human memory: A proposed system and its control processes. In K.W. Spence, & J.W. Spence (Eds.), *The Psychology of Learning and Motivation Volume 2* (pp. 89-195). London: Academic Press.

Backman, L, Small, B.J., & Wahlin, A. (2001). Aging and memory: Cognitive and biological perspectives. In Birren, J.E., Schaie, K.W. (Eds.), *Handbook of the Psychology of Aging, 5th Edition* (pp. 349-377). San Diego, CA: Academic Press.

Backman, L., Ginovart, N., Dixon, R.A., Wahlin, T-B.R., Halldin, C., & Farde, L. (2000). Age-related cognitive deficits mediated by changes in the striatal dopamine system. *American Journal of Psychiatry*, *157(4)*, 635-637.

Baddeley, A.D. (1986). *Working Memory*. Oxford: Clarendon Press.

Baddeley, A.D., & Hitch, G. (1974). Working memory. In Bower, G.H. (Ed.), *The Psychology of Learning and Motivation: Advances in Research and Theory, Volume 8* (pp. 47-89). New York: Academic Press.

Badre, D., & Wagner, A.D. (2005). Frontal lobe mechanisms that resolve proactive interference. *Cerebral Cortex*, *15(12)*, 2003-2012.

Bailey, L.L., & Hansson, R.O., (1995). Psychological obstacles to job or career change in late-life. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 50* (6), 280-288.

Band, G.P.H., Ridderinkhof, K.R., & Segalowitz, S. (2002). Explaining neurocognitive aging: Is one factor enough? *Brain and Cognition*, *49(3)*, 259-267.

Barusch, A., Rogers, A., & Abu-Bader, S.H. (1999). Depressive symptoms in the frail elderly: physical and psycho-social correlates. *International Journal of Aging and Human Development*, *49 (2)*, 107-125.

Baxendale, S. (1995). The hippocampus: functional and structural correlations. *Seizure*, *4*, 105-117.

Beaulieu, C., & Colonnier, M. (1988). Richness of environment affects the number of contacts formed by boutons containing flat vesicles but does not alter the number of these boutons per neuron. *Journal of Comparitive Neurology*, *274*, 347-356.

Bechara, A., Tranel, D., & Damasio, H. (2000). Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. *Brain*, *123(11)*, 2189-2202.

Bellebaum, C., & Daum, I. (2004). Effects of age and awareness on eyeblink conditional discrimination learning. *Behavioral Neuroscience*, *118(6)*, 1157-1165.

Birren, J.E., & Schroots, J.J.F. (2001). History of Geropyschology. In Birren, J.E., & Schaie, K.W. (Eds.), *Handbook of the Psychology of Aging, 5th Edition* (pp. 3-28). San Diego, CA: Academic Press.

Bosman, E.A. (1993). Age related differences in the motoric aspects of transcription typing skill. *Psychology and Aging*, *8*, 87-102.

Bosworth, H.B., & Siegler, I.C. (2002). Terminal change in cognitive function: an updated review of longitudinal studies. *Experimental Aging Research*, *28*, 299-315.

Bottwinick, J., Robbin, J.S. & Brinley J.F. (1960). Age differences in card sorting performance in relation to task difficulty, task set, and practice. *Journal of Experimental Psychology*, *59*, 10-18.

Botwinick, J., & Birren, J.E. (1963). Mental Abilities and Psychomotor Responses in Healthy Aged Men. In Birren, J.E., Butler, R.N., Greenhouse, S.W., Sokoloff, L., & Yarrow, M.R. (Eds.), *Human Aging: A Biological and Behavioral Study* (pp. 97-108). Bethesda, MD: US Dept of Health, Education, & Welfare.

Bowles, R.P., & Salthouse, T.M.A. (2003). Assessing the age-related effects of proactive interference on working memory tasks using the Rasch model. *Psychology and Aging*, *18*, 608-615.

Brinley, J.F. (1965). Cognitive sest, speed and accuracy of performance in the elderly. In Welford, A.T., & Birren, J.E. (Eds.), *Behavior, Aging and The Nervous System* (Pps. 114-149). Springfield, IL: Thomas.

Budson, A.E., Dodson, C.S., Vatner, J.M., Daffner, K.R., Black, P.M., & Schachter, D.L. (2005). Metacognition and false recognition in patients with frontal lobe lesions: the distinctiveness heuristic. *Neuropsychologia*, *43*, 860-871.

Buzsaki, G. (1989). Two stage model of memory-trace formation: A role for 'noisy' brain states. *Neuroscience*, *31(3)*, 551-570.

Cacioppo, J.T., Petty, R.E., & Kao, C.F. (1984). The efficient assessment of need for cognition. *Journal of Personality Assessment*, *48*, 306-307.

Cattell, R.B. (1963). Theories of fluid and crystallized intelligence: A critical experiment. *Journal of Educational Psychology*, *54(1)*, 1-22.

Cattell, R.B., & Cattell, A.K.S. (1960). *The Individual or Group Culture Fair Intelligence Test*. Champaign, Il: IPAT.

Cerella, J. (1985). Information processing rates in the elderly. *Psychological Bulletin*, *98(1)*, 67-83.

Chapman, G.B. & Robbins, S.J. (1990). Cue interaction in human contingency judgment. *Memory and Cognition*, *18*, 537–545.

Charness, N. (1981). Visual short-term memory and aging in chess players. *Journal of Gerontology*, *36*, 615-619.

Charness, N. (1991). Expertise in chess: The balance between knowledge and search. In Ericsson, K.A., & Smith, J. (Eds.). *Toward a General Theory of Expertise: Prospects and Limits* (pp. 39-63). New York: Cambridge University Press.

Chaytor, N., & Schmitter-Edgecombe, M. (2004). Working memory and aging: A cross-sectional and longitudinal analysis using a self-ordered pointing task. *Journal of the International Neuropsychological Society*, *10(4)*, 489-503.

Chodosh, J., Seeman, T.E., Keeler, E., Sewall, A., Hirsch, S.H., Guralnik, J.M., & Reuben, D.B. (2004). Cognitive decline in high-functioning older persons is associated with an increased risk of hospitalization. *Journal of the American Geriatrics Society*, *52(9)*, 1456-1462.

Clay, H.M. (1954). Changes in performance with age on similar tasks of varying complexity. *British Journal of Psychology*, *45*, 7-13.

Cohen, D. (1986). Psychopathological perspectives: Differential diagnosis of Alzheimer's disease and related disorders. In Poon, L.W., & Crook, T. (Eds.), *Handbook for Clinical Memory Assessment of Older Adults* (pp. 81-88). Washington DC: American Psychological Association.

Cohen, N.J., Ryan, J., Hunt, C., Romine, L., Wszalek, T., & Nash, C. (1999). Hippocampal system and declarative (relational) memory: Summarising the data from functional neuroimaging studies. *Hippocampus*, *9*, 83-98.

Craik, F. I. M. (1986). A functional account of age differences in memory. In F. Klix & H. Hagendorf (Eds.), *Human Memory and Cognitive Capabilities* (pp. 409-422). New York: Elsevier Science Publishers.

Craik, F.I.M., & Jennings, J.M., (1992). Human memory. In Craik, F.I.M. & Salthouse, T.A., (Eds.), *The Handbook of Aging and Cognition* (pp. 51-110). Hillsdale: Lawrence Erlbaum.

Crawford, J. R., Bryan, J., Luszcz, M. A., Obonsawin, M.C., & Stewart, L. (2000). The executive decline hypothesis of cognitive aging: Do executive deficits qualify as differential deficits and do they mediate age-related memory decline? *Aging, Neuropsychology and Cognition*, *7*, 9–31.

Cunningham, W.R., & Owens, W.A. (1983). The Iowa State study of the adult development of intellectual abilities. In Schaie, K.W. (Ed.), *Longitudinal Studies of Adult Psychological Development* (pp. 64-135). New York: Guilford Press.

Damasio, A.R. (1994). The brain binds entities and events by multiregional activation from convergence zones. In Gutfreund, H., & Toulouse, G (Eds.), *Biology and Computation: A Physicist's Choice* (pp. 749-758). River Edge, NJ: World Scientific Publishing Co.

Daneman, M., & Carpenter, P.A. (1980). Individual differences in working memory and reading. *Journal of Verbal Learning and Verbal Behavior*, *19*, 450-466.

Darby, R.J., & Maylor, E.A. (1998). Effects of the relationship between background and prospective memory task requirements on age differences in prospective memory. Poster presented at the Seventh Cognitive Aging Conference, April, Atlanta, Georgia.

Daum, I., Channon, S., Pokey. C.A., & Gray, J.A. (1991). Classical conditioning after temporal lobe lesions in man: impairment in conditional discrimination. *Behavioral Neuroscience*, *105*, 396-408.

De Houwer, J., & Beckers, T. (2002). A review of recent developments in research and theories on human contingency learning. *Quarterly Journal of Experimental Psychology*, *55B(4)*, 289-310.

De Ribaupierre, A. (2002). Working memory and attentional processes across the lifespan. In P.Graf & N. Ohta (Eds.), *Lifespan Development of Human Memory* (pp. 59-80). Cambridge, MA: The MIT Press.

Deary, I.J., Wright, A.F., Harris, S.E., Whalley, L.J., & Starr, J.M. (2004). Searching for genetic influences on normal cognitive ageing. *Trends in Cognitive Science*, *8(4)*, 178-184.

Dempster F.N., & Corkhill A. (1999). Neo-interference research and the development of intelligence. In Anderson M. (Ed.), *The Development of Intelligence* (pp. 215-243). Hillsdale, NJ: Psychology Press.

Desgranges B., Baron J.C., & Eustache F. (1998). The functional neuroanatomy of episodic memory: the role of the frontal lobes, the hippocampal formation, and other areas. *Neuroimage*, *8(2)*, 198-213.

Diamond, M.C., Johnson, R.E., Protti, A.M. Ott, C., & Kajisa, L. (1985). Plasticity in the 904-day-old male rat cerebral cortex. *Experimental Neurology*, *87*, 309-317.

Dickinson, A. (1994). Instrumental Conditioning. In N.J. Mackintosh (Ed.), *Animal Cognition and Learning* (pp. 45-79). London: Academic Press

Dickinson, A. (1980). *Contemporary Learning Theory*. Cambridge: Cambridge University Press.

Dickinson, A. (2001). The 28th Bartlett Memorial Lecture. Causal Learning: An Associative Analysis. *Quarterly Journal of Experimental Psychology*, *54B*, 3-25

Dickinson, A., & Shanks, D. (1985). Animal conditioning and human causality judgment. In Nilsson, L.G., & Archer, T. (Eds.), *Perspectives on Learning and Memory. Series in Comparative Cognition and Neuroscience* (pp. 167-191). Umea, Sweden: University of Umea.

Dickinson, A., Shanks, D.R., & Evendon, J.L. (1984). Judgement of Act Outcome Contingency: the Role of Selective Attribution. *Quarterly Journal of Experimental Psychology*, *49B*, 97-110.

Dimitrov, M., Granetz, J., Peterson, M., Hollnagel, C., Alexander, G., & Grafman, J. (1999). Associative Learning Impairments in Patients with Frontal Lobe Damage. *Brain and Cognition, 41*, 213-230.

Duff, S.J., & Hampson, E. (2000). A Beneficial Effect of Estrogen on Working Memory in Post Menopausal Women Taking Hormone Replacement Therapy. *Hormones & Behaviour*, *38*, 262-276.

Duncan, J. (1995). Attention, intelligence, and the frontal lobes. In Gazzaniga, M.S. (Ed.). *The Cognitive Neurosciences* (pp. 721-733). Cambridge, MA: MIT Press.

Duncan, J. (2005). Frontal lobe function and general intelligence: why it matters. *Cortex, 41*, 215-217.

Duncan, J., & Owen, A.M. (2000). Common regions of the frontal lobe recruited by diverse cognitive demands. *Trends in Neurosciences*, *23*, 475-483.

Duncan, J., Burgess, P., & Emslie, H. (1995). Fluid intelligence after frontal lobe lesions. *Neuropsychologia, 33*, 261-268.

Earles, J.L., & Coon, V.E. (1994). Adult age differences in long term memory for performed activities. *Journals of Gerontology: Psychological Sciences*, *49(1)*, 32-34.

Ebbinghaus, H. (1913). *Memory: A Contribution to Experimental Psychology*. New York: Teacher's College Press.

Ebert, D.A., & Heckerling, P.S. (1998). Communication disabilities among medical inpatients. *New England Journal of Medicine*, *339 (4)*, 272-273.

Eichenbaum, H., Otto, T., & Cohen, N.J. (1994). Two functional components of the hippocampal memory system. *Behavioural and Brain Sciences*, *17*, 449-518.

Ellis, R., & Humphreys, G. (1999). *Connectionist Psychology*. Hove: Psychology Press.

Erixon-Lindroth, N., Farde, L., Wahlin, T.R., Sovago, J., Halldin, C., & Backman, L. (2005). The role of the striatal dopamine transporter in cognitive aging. *Psychiatry Research: Neuroimaging*, *138*, 1-12.

Estes, W.K. (1950). Towards a statistical theory of learning. *Psychological Review*, *57*, 94-107.

Estes, W.K. (1959). The statistical approach to learning theory. In Koch, S. (Ed.), *Psychology: A Study of Science, Volume 2* (pp. 380-491). New York: McGraw-Hill.

Flynn, J.R. (1984). The mean IQ of Americans: Massive gains 1932-1978. *Psychological Bulletin*, *95*, 29-51.

Flynn, J.R. (1987). Massive IQ gains in 14 nations: What IQ tests really measure. *Psychological Bulletin*, *101*, 171-191.

Foster, J.C., & Taylor, G.A. (1920). The applicability of mental tests to persons over 50. *Journal of Applied Psychology*, *4*, 39-58.

Fox, S., Wolfson, S., & Ranck, J. (1986). Hippocampal theta rhythm and the firing of neurons in walking and urethane anesthetized rats. *Experimental Brain Research*, *62*, 495–508.

Fozard, J.L., & Gordon-Salant, S. (2001). Changes in hearing and vision with aging. In Birren, J.E., Schaie, K.W. (Eds.), *Handbook of the Psychology of Aging, 5th Edition* (pp. 241-266). San Diego, CA: Academic Press.

Francis, S., Rolls, E.T., Bowtell, R., McGlone, F., O'Doherty, J., Browning, A., Clare, S., & Smith, E. (1999). The representation of pleasant touch in the brain and its relationship with taste and olfactory areas. *Neuroreport: For Rapid Communication of Neuroscience Research*, *10(3)*, 453-459

Gabrielli, J.D.E., McGlinchey-Berroth, R., Carillo, M., Gluck, M.A., Cermack, L., & Disterhoft, J. (1995). Intact eye-blink classical conditioning in amnesia. *Behavioural Neuroscience*, *109*, 819-827.

Gabrielli, J.D.E. (1998). Cognitive neuroscience of human memory. *Annual Review of Psychology*, *49*, 87-105.

Glisky, E.L., Rubin, S.R., & Davidson, P.S.R. (2001). Source memory in older adults: An encoding or retrieval problem? *Journal of Experimental Psychology: Learning, Memory, & Cognition*, *27(5)*, 1131-1146.

Gluck, M., & Myers, C.E. (1993). Hippocampal Mediation of Stimulus Representation: A Computational Theory. *Hippocampus*, *3 (4)*, 491-516.

Gluck, M., & Myers, C.E. (1996). Integrating behavioral and physiological models of hippocampal function. *Hippocampus*, *6*, 643-653.

Gluck, M., & Myers, C.E. (2001). *Gateway to Memory: An Introduction to Neural Network Models of the Hippocampus and Learning*. Cambridge, MA: MIT Press.

Goldberg, E., & Bilder, R.M. (1987). The frontal lobes and hierarchical organization of cognitive control. In E. Perecman (Ed.), *Frontal Lobes Revisited* (pp. 159-187). New York: The IRBN Press.

Granick, S., Kleban, M.H., & Weiss, D. (1976). Relationships between hearing loss and cognition in normally hearing aged persons. *Journal of Gerontology*, *31(4)*, 434-440.

Gunning-Dixon, F.M., & Raz, N. (2003). Neuroanatomical correlates of selected executive functions in middle-aged and older adults: A prospective MRI study. *Neuropsychologia*, *41(14)*, 1929-1941.

Guttman, N., & Kalish, H. (1956). Discriminability and stimulus generalisation. *Journal of Experimental Psychology*, *51*, 79-88.

Hanson, H.M. (1959). Effects of discrimination training on stimulus generalization. *Journal of Experimental Psychology*, *58*, 321-334.

Hartley, A.A., (1992). Attention. In Craik, F.I.M. & Salthouse, T.A. (Eds.), *The Handbook of Aging and Cognition* (pp. 3-49). Hillsdale: Lawrence Erlbaum.

Hartman, A., Pickering, R. M., & Wilson, B. A. (1992). Is there a central executive deficit after severe head injury? *Clinical Rehabilitation*, *6(2)*, 133-140.

Hasher, L., & Zacks, R.T. (1988). Working memory, comprehension, and aging: A review and a new view. In G.H. Bower (Ed.), *The Psychology of Learning and Motivation* (pp. 193-225). San Diego, CA: Academic Press.

Hasher, L., Stoltzfus, E.R., Zacks, R.T., & Rympa, B. (1991). Age and inhibition. *Journal of Experimental Psychology: Learning, Memory, & Cognition*, *17(1)*, 163-169.

Hasselmo, M. (1995). Neuromodulation and cortical function: Modeling the physiological basis of behavior. *Behavioural Brain Research*, *67*, 1-27.

Hasselmo, M., & Schnell, E. (1994). Laminar selectivity of the cholinergic suppression of synaptic transmission in rat hippocampal region CA1: Computational modeling and brain slice physiology. *Journal of Neuroscience*, *14(6)*, 3898-3914.

Hasselmo, M., Schnell, E., & Barkai, E. (1995). Dynamics of learning and recall at excitatory recurrent synapses and cholinergic modulation in rat hippocampal region CA3. *Journal of Neuroscience*, *15(7)*, 5249-5262.

Hebb, D. (1949). *The Organization of Behaviour*. New York: Wiley.

Hebert, L.E., Scherr, P.A., Bennett, D.A., Bienias, J.L., Wilson, R.S., Morris, M.C., & Evans, D.A. (2004). Blood Pressure and Late-Life Cognitive Function Change: A Biracial Longitudinal Population Study. *Neurology*, *62(11)*, 2021-2024.

Hedden, T., & Park, D.C. (2001). Aging and interference in verbal working memory. *Psychology and Aging*, *16*, 666-681.

Hedden, T., & Park, D.C. (2003). Contributions of source and inhibitory mechanisms to age related retroactive interference in verbal learning memory. *Journal of Experimental Psychology: General*, *132(1)*, 93-112.

Heim, A. (1968). *The AH4 Test*. Windsor: NFER Nelson.

Henke, K., Weber, B., Kneifel, S., Wiesler, H.G., & Buck, A. (1999). Human hippocampus associates information in memory. *Proceedings of the National Academy of Sciences U.S.A.*, *96*, 5884-5889.

Henkel, L.A., Johnson, M.K., & De Leonardis, D.M. (1998). Aging and source monitoring: Cognitive processes and neuropsychological correlates, *Journal of Experimental Psychology: General*, *127 (3)*, 251-268.

Henry, J.D., MacLeod, M.S., Phillips, L.H., & Crawford, J.R. (2004). A meta-analytic review of prospective memory and aging. *Psychology and Aging*, *19(1)*, 27-39.

Honig, W.K. (1962). Prediction of preference, transposition, and transposition reversal from the generalisation gradient. *Journal of Experimental Psychology*, *64*, 239-248.

Hopkins, R.O., Myers, C.E., Shohamy, D., Grossman, S., & Gluck, M. (2004). Impaired probabilistic category learning in hypoxic subjects with hippocampal damage. *Neuropsychologia*, *42*, 524-535.

Horn, J.L. (1989). Models for Intelligence. In Linn, R. (Ed.), *Intelligence: Measurement, Theory and Public Policy* (pp. 29-73). Urbana, IL: University of Illinois Press.

Horn, J.L., & Cattell, R.B. (1967). Age differences in fluid and crystallized intelligence. *Acta Psychologica*, *26*, 107-129.

Horn, J.L., & Mazanuga, H. (2000). New directions for research into aging and intelligence. In Perfect, T.J., & Maylor, E.A. (Eds.), *Models of Cognitive Aging* (pp. 125-159). Oxford: Oxford University Press.

Jacobs, B., Schall, M., & Scheibel, A.B. (1993). A quantitative dendritic analysis of Wernicke's area in humans II. Gender, hemispheric, and environmental factors. *Journal of Comparitive Neurology*, *327*, 97-111.

Jarrard, L.E. (1993). On the role of the hippocampus in learning and memory in the rat. *Behavioral and Neural Biology*, *60*, 9-26.

Jelicic, M., & Kempen, G.I.J.M. (1999). Effect of self rated health on cognitive performance in community dwelling elderly. *Educational Gerontology*, *25(1)*, 13-17.

Johnson, M.K., Hashtroudi, S., & Lindsay, D.S. (1993). Source monitoring. *Psychological Bulletin*, *114(1)*, 3-28.

Kamin, L.J. (1969). Predictability, surprise, attention and conditioning. In Campbell, B.A., & Church, R.M., *Punishment and Aversive Behaviour* (pp. 279-296). New York: Appleton-Century-Crofts.

Kane, M.J., Hasher, L., & Stoltzfus, E.R. (1994). Inhibitory attentional mechanisms and aging. *Psychology & Aging*, *9(1)*, 103-112.

Kehoe, E.J. (1988). A layered network model of associative learning: Learning to learn and configuration. *Psychological Review*, *95(4)*, 411-433.

Kinsbourne, M. (1980). Attentional dysfunctions and the elderly: Theoretical models and research perspectives. In Poon, L.W., Fozard, J.L., Cermak, L.S., Arenberg, D., Thompson, L.W. (Eds.), *New Directions in Memory and Aging* (pp. 113-129). Hillsdale, NJ: Lawrence Erlbaum Associates.

Klatsky, R.L. (1988). Theories of information processing and theories of aging. In Light, L.L., & Burke, D.M. (Eds.), *Language, Memory, and Aging*. New York: Cambridge University Press.

Kleemeir, R.W. (1962). Intellectual changes in the senium. *Proceedings of the Social Statistics Section of the American Statistical Association*, *1*, 290-295.

Kliegal, M., Zimprich, D., & Rott, C. (2004). Lifelong intellectual activities mediate the predictive effect of early education on cognitive impairment in centarians: a retrospective study. *Aging and Mental Health*, *8(5)*, 430-437.

Kliegel, M., McDaniel, M.A., & Einstein, G.O. (2000). Plan formation, retention, and execution in prospective memory: A new approach and age-related effects. *Memory and Cognition*, *28(4)*, 1041-1049.

Knowlton, B.J., Squire, L.R., & Gluck, M.A. (1994). Probabilistic category learning in amnesia. *Learning and Memory*, *1*, 106-120.

Konorski, J. (1948). *Conditioned Reflexes and Neuron Organisation*. Cambridge: Cambridge University Press.

Konorski, J. (1967). *Integrative Activity of the Brain*. Chicago: Chicago University Press.

Kringelbach, M.L., O'Doherty, J., Rolls, E.T., & Andrews, C. (2003). Activation of the human orbitofrontal cortex to a liquid food stimulus is correlated with its subjective pleasantness. *Cerebral Cortex*, *13(10)*, 1064-1071.

Kruschke, J.K. (1992). ALCOVE: An exemplar based connectionist model of category learning. *Psychological Review*, *99 (1)*, 22-44.

Lamar, M., & Resnick, S.M. (2004). Aging and prefrontal functions: Dissociating orbitofrontal and dorsolateral abilities. *Neurobiology of Aging*, *25(4)*, 553-558.

Lashley, K.S., & Wade, M. (1946). The Pavlovian theory of generalization. *Psychological Review, 53*, 72-87.

LaVoie, D.J., Willoughby, L., & Faulkner, K. (2006). Frontal lobe dysfunction and false memory susceptibility in older adults. *Experimental Aging Research*, *32*, 1-21.

Lawrence, D.H. (1952). The transfer of a discrimination along a continuum. *Journal of Comparative Psychology, 45,* 511-516.

LePelley, M.E. (2004). The role of associative history in models of associative learning: a selective review and a hybrid model. *Quarterly Journal of Experimental Psychology*, *57B(3)*, 193-243.

Light, L.L. (1991). Memory and aging: Four hypotheses in search of data. *Annual Review of Psychology*, *42*, 333-376.

Light, L.L., & La Voie, D. (1993). Direct and indirect measures of memory in old age. In P. Graf, & Masson, M.E.J. (Eds.), *Implicit memory: New Directions in Cognition, Development, and Neuropsychology* (pp.207-230). Hillsdale, NJ: Lawrence Erlbaum Associates.

LoLordo, V.M., & Fairless, J. (1985). Pavlovian conditioned inhibition: The literature since 1969. In Miller, R.R., and Spears, N.E. (Eds.), *Information Processing in Animals: Conditioned Inhibition* (pp1-50). Hillsdale, NJ: Lawrence Erlbaum Associates.

Luo, J., & Niki, K. (2003). Function of hippocampus in 'insight' of problem solving. *Hippocampus*, *13(3)*, 316-323.

Mackintosh, N.J. (1974). *The Psychology of Animal Learning*. New York: Academic Press.

Mackintosh, N.J. (1975). A theory of attention: variation in the associability of a stimulus with reinforcement. *Psychological Review*, *82*, 276-298.

Mackintosh, N.J. (2003). Pavlov and associationism. *The Spanish Journal of Psychology*, *6*, 177-184.

Mackintosh, N.J., & Little, L. (1970). An analysis of transfer along a continuum. *Canadian Journal of Psychology, 24,* 362-369.

MacQuarrie, T.W. (1953). *MacQuarrie Test for Mechanical Ability*. Monterey: McGraw-Hill.

Madden, D.J. (2001). Age-related declines in motor control. In Birren, J.E., Schaie, K.W. (Eds.), *Handbook of the Psychology of Aging, 5th Edition* (pp. 288-312). San Diego, CA: Academic Press.

Maddox, G, & Douglas, E. (1973). Aging and individual differences. *Journal of Gerontology, 29*, 555-563.

Mani, T.M., Bedwell, J.S., & Miller, L.S. (2005). Age-related decrements in performance on a brief continuous performance test. *Archives of Clinical Neuropsychology*, *20(5)*, 575-586.

Marr, D. (1970). A theory for cerebral neocortex. *Proceedings of the Royal Society of London, B176(43)*, 161-234.

Marr, D. (1971). Simple memory: A theory for archicortex. *Proceedings of the Royal Society of London, B262(841)*, 23-81.

Martin, M., Kliegel, M., & McDaniel, M.A. (2003). The involvement of executive functions in prospective memory performance of adults. *International Journal of Psychology*, *38(4)*, 195-206.

Mather, M., & Johnson, M.K. (2003). Affective review and schema reliance in memory in older and younger adults. *American Journal of Psychology*, *116(2)*, 169-189.

Maylor, E.A. (1998). Changes in event based prospective memory across adulthood. *Aging, Neuropsychology, and Cognition*, *5*, 107-128.

Maylor, E.A., Darby, R.J., Logie, R.H., Della Sala, S., & Smith, G. (2002). Prospective memory across the lifespan. In P. Graf, & N. Ohta (Eds.), *Lifespan Development of Human Memory* (pp.235-256). Cambridge, MA: MIT Press.

McCarthy, F.M., Sellers, A.H., & Burns, W.J. (2003). Prediction of IQ in the Mayo adult normative sample using multiple methods. *Journal of Clinical Psychology*, *59*, 457-463.

McClearn, G.E., & Vogler, G.P. (2001). The genetics of behavioral aging. In Birren, J.E., Schaie, K.W. (Eds.), *Handbook of the Psychology of Aging, 5th Edition* (pp. 109-134). San Diego, CA: Academic Press.

McClearn, G.E. (2002). Genetics of behavioural aging: animal models and the human condition. *Experimental Aging Research*, *28*, 453-476.

McCormack, T, Brown, G.D.A., Maylor, E.A., Darby, R.J., & Green, D. (1999). Developmental changes in time estimation: Comparing childhood and old age. *Developmental Psychology*, *35*, 1143-1155.

McCurry, S.M., Gibbons, L.E., Uomoto, J.M., Thomson, M.L., Graves, A.B., Edland, S.D., Bowen, J., McCormick, W.C., & Larson, E.B. (2001). Neuropsychological test performance in a cognitively intact sample of older Japanese American adults. Archives of Clinical Neuropsychology, *16(5)*, 447-459.

McDaniel, M.A., Glisky, E.L., Guynn, M.J., & Routhieaux, B.C. (1999). Prospective memory: A neuropsychological study. *Neuropsychology*, *13(1)*, 103-110.

McDonald, L., & Stuart-Hamilton, I. (2003). Egocentrism in older adults: Piaget's three mountains task revisited. *Educational Gerontology*, *29*, 417-425.

Mednick, S.A., & Freedman, J.L. (1960). Stimulus generalization. *Psychological Bulletin*, *57*, 169-200.

Mell, T., Heekeren, H.R., Marschner, A., Wartenburger, I., Villringer A., & Reischies, F.M. (2005). Effect of aging on stimulus-reward association learning. *Neuropsychologia*, *43*, 554-563.

Milham, M.P., Erickson, K.I., Banich, M.T., Kramer, A.F., Webb, A., Wszalek, T., & Cohen, N.J. (2002). Attentional control in the aging brain: Insights from an fMRI study of the Stroop task. *Brain and Cognition*, *49*, 277-296.

Mishkin, M. (1978). Memory in monkeys severely impaired by combined but not by separate removal of amygdala and hippocampus. *Nature*, *273*, 297-298

Mitchell, C.J., & Lovibond, P.F. (2002). Backward and forward blocking in human electrodermal conditioning: Blocking requires an assumption of outcome additivity. *Quarterly Journal of Experimental Psychology*, *55B*, 311-329.

Miyake A., Friedman N.P., Emerson M.J., Witzki A.H., Howerter A., & Wager T.D. (2000). The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: a latent variable analysis. *Cognitive Psychology*, *41(1)*, 49-100.

Miyake, A., & Shah, P. (1999). *Models of Working Memory: Mechanisms of Active Maintenance and Executive Control*. New York: Cambridge University Press.

Musch, J., & Ehrenberg, K. (2002). Probability misjudgement, cognitive ability, and belief in the paranormal. *British Journal of Psychology*, *93*, 169-177.

Mutter, S.A., & Williams, T.W. (2004). Aging and the detection of contingency in causal learning. *Psychology and Aging*, *19(1)*, 13-26.

Myers, C.E., Ermita, B.R., Harris, K., Hasselmo, M., Soloman, P., & Gluck, M.A. (1996). A computational model of septohippocampal activity in classical eyeblink conditioning. *Neurobiology of Learning and Memory*, *66*, 51-66.

Myers, C.E., Gluck, M., & Granger, R. (1995). Dissociation of hippocampal and entorhinal function in associative learning: a computational approach. *Psychobiology*, *23 (2)*, 116-138.

Myerson, J., Adams, D.R., Hale, S., & Jenkins, L. (2003). Analysis of group differences in processing speed: Brinley plots, Q-Q plots and other conspiracies. *Psychonomic Bulletin & Review*, *10(1)*, 224-237.

Naidu, A., Xu, Q., Catalano, R., & Cordell, B. (2002). Secretion of apolipoprotein E by brain glia requires protein prenylation and is suppressed by statins. *Brain Research*, *958*, 100-111.

Naveh-Benjamin, M., & Craik, F.I.M., (1995). Memory for context and its use in item memory: comparisons of younger and older people. *Psychology and Aging*, *10(2)*, 284-293.

Naveh-Benjamin, M. (2000). Adult age differences in memory performance: tests of an associative deficit hypothesis. *Journal of Experimental Psychology: Learning, Memory & Cognition*, *26(5)*, 1170-1187.

Naveh-Benjamin, M., Craik, F.I.M., & Ben-Shaul, L. (2002). Age related differences in cued-recall: Effects of support at encoding and retrieval. *Aging, Neuropsychology, and Cognition*, *9(4)*, 278-287.

Naveh-Benjamin, M., Hussain, Z., Guez, J., & Bar-On, M. (2003). Adult age differences in episodic memory: further support for an associative deficit hypothesis. *Journal of Experimental Psychology: Learning, Memory & Cognition*, *29(5)*, 826-837.

Naveh-Benjamin, M., Guez, J., Kilb, J., & Reedy, S. (2004). The associative memory deficit of older adults: further support using face-name associations. *Psychology and Aging*, *19(3)*, 541-546.

Novick, L.R., & Holyoak, K.J. (1991). Mathematical problem solving by analogy. *Journal of Experimental Psychology: Learning, Memory & Cognition, 17,* 398-415.

Novick, L.R., & Sherman, S.J. (2003). On the nature of insight solutions: evidence from skill differences in anagram solution. *Quarterly Journal of Experimental Psychology, 56A,* 351-382.

OFFICE OF NATIONAL STATISTICS, (2002). *Census 2001: Population Report, Demographics*. <http://www.statistics.gov.uk/census2001/demographic_uk.asp>, accessed 3/10/2002.

Park D.C., Hertzog C., Kidder D.P., Morrell R.W., & Mayhorn C.B. (1997). Effect of age on event-based and time-based prospective memory. *Psychology of Aging*, *12(2)*, 314-27.

Parkin, A.J., & Java, R.I. (1999). Deterioration of frontal lobe function in normal aging: influences of fluid intelligence versus perceptual speed. *Neuropsychology*, *13 (4)*, 539-545.

Parkin, A.J., & Java, R.I. (2000). Determinants of age related memory loss. In T.J. Perfect, & E.A. Maylor, *Models of Cognitive Ageing* (pp. 188-203). Oxford: Oxford University Press.

Pavlov, I.P. (1927). *Conditioned Reflexes*. New York: Oxford University Press.

Pearce, J.M. (1987). A model for stimulus generalization in Pavlovian conditioning. *Psychological Review*, *94*, 61-73.

Pearce, J.M. (1994). Similarity and discrimination: a selective review and a connectionist model. *Psychological Review*, *101 (4)*, 587-607.

Pearce, J.M. (2002). Evaluation and development of a connectionist theory of configural learning. *Animal Learning & Behavior*, *30*, 73-95.

Pearce, J.M., Adam, J., Wilson, P.N., & Darby, R.J. (1992). Effects of Discrimination Training on responding During a Compound Conditioned Stimulus. *Journal of Experimental Psychology: Animal Behavior Processes*, *18*, 379-386

Pearce, J.M., & Hall, G. (1980). A model for Pavlovian conditioning: variations in the effectiveness of conditioned but not of unconditioned stimuli. *Psychological Review*, *87*, 532-552.

Pearce, J.M., & Redhead, E.S. (1995). Supernormal conditioning. *Journal of Experimental Psychology: Animal Behavior Processes*, *21*, 155-165.

Perfect, T.J. (1994). What can Brinley plots tell us about cognitive aging? *Journal of Gerontology: Psychological Sciences*, *49*, 60-64.

Perfect, T. J., & Maylor, E.A. (2001). Rejecting the dull hypothesis: the relation between method and theory in cognitive aging research. In T.J. Perfect, & E.A. Maylor, *Models of Cognitive Ageing* (pp. 1-18). Oxford: Oxford University Press.

Perret, E. (1974). The left frontal lobe of man and the suppression of habitual responses in verbal categorical behaviour. *Neuropsychologia*, *12(3)*, 323-30.

Petrides, M., & Milner, B. (1982). Deficits in self-ordered tasks after frontal- and temporal-lobe lesions in man. *Neuropsychologia*, *20*, 249-262.

Piaget, J., & Inhelder, B. (1956). *The Child's Conception of Space*, New York: Norton.

Pilpel, D., Carmel, S., & Galinsky, D. (1988). Self-rated health among the elderly. A comparative analysis of health status measures, leisure activities and social contacts in age/sex groups. *Comprehensive Gerontology, B*, *2(3)*, 110-116.

Plomin, R., & Spinath, F.M. (2002). Genetics and general cognitive ability. *Trends in Cognitive Science*, *6*, 169-176.

Plude, D.J., Benaderet, T., & Herrmann, D.J. (2001). Aging, memory assessment and self-reported function. *International Journal of Cognitive Technology*, *6(1)*, 38-40.

Pushkar, D., Arbuckle, T., Conway, M., Chaikelson, J., & Maag, U. (1997). Everyday activity parameters and competence in older adults. *Psychology and Aging*, *12 (4)*, 600-609.

Rabbitt, P.M.A. (1993). Does it all go together when it goes? The nineteenth Bartlett Memorial Lecture. *Quarterly Journal of Experimental Psychology*, *46A(3)*, 385-434.

Rabbitt, P.M.A. (2005). Frontal brain changes and cognitive performance in old age. *Cortex*, *41*, 238-240.

Rabbitt, P.M.A., Chetwynd, A, & McInnes, L. (2003). Do clever brains age more slowly? Further exploration of a nun result. *British Journal of Psychology*, *94(1)*, 63-71.

Rabbitt, P.M.A., & Lowe, C. (2000). Patterns of cognitive ageing. *Psychological Research*, *63*, 380-316.

Rabbitt, P.M.A., Lowe, C., & Shilling, V. (2001). Frontal tests and models of cognitive ageing. *European Journal of Experimental Psychology*, *13*, 5-28.

Rabbitt, P.M.A., McInnes, L., Diggle, P., Holland, F., Bent, N., Abson, V., Pendleton, N., & Horan, M. (2004). The University of Manchester longitudinal study of cognition in normal healthy old age, 1983 through 2003. *Aging Neuropsychology and Cognition*, *11(2-3)*, 245-279.

Ratcliff, R., Spieler, D., & McKoon, G. (2000). Explicitly modelling the effects of aging on response times. *Psychonomic Bulletin & Review*, *7*, 1-25.

Ratcliff, R., Thapar, A., & McKoon, G. (2001). The effects of aging on reaction time in a signal detection task. *Psychology and Aging*, *16(2)*, 323-341.

Ratcliff, R., Spieler, D., & McKoon, G. (2004). Analysis of group differences in processing speed: Where are the models of processing? *Psychonomic Bulletin & Review*, *11*, 755-769.

Raven, J. (1982). *The Mill Hill Vocabulary Scale*. Oxford: Oxford Psychologists Press.

Raz, N. (2000). Aging of the brain and its impact on cognitive performance: integrations of structural and functional findings. In Craik, F.I.M. & Salthouse, T.A., (Eds). *The Handbook of Aging and Cognition, 2nd Edition* (pp. 1-90). Mahwah, NJ: Lawrence Erlbaum.

Raz, N., Gunning-Dixon, F.M., Head, D. P., Dupuis, J.H., & Acker, J.D. (1998). Neuroanatomical correlates of cognitive aging: Evidence from structural MRI. *Neuropsychology*, *12*, 95-106.

Reberg, D., & Leclerc, R. (1977). A feature positive effect in conditioned suppression. *Animal Learning and Behaviour*, *5*, 143-147.

Redhead, E.S., & Pearce, J.M., (1995). Similarity and Discrimination Learning. *Quarterly Journal of Experimental Psychology, 48B (1),* 46-66.

Rescorla, R.A. (1973). Evidence for a "unique cue" stimulus account of configural conditioning. *Journal of Comparative and Physiological Psychology*, *85*, 331-338.

Rescorla, R.A. (1979). Conditioned inhibition and extinction. In Dickinson, A., & Boakes, R.A. (Eds), *Mechanisms of Learning and Motivation* (pp. 83-110). Hillsdale, NJ: Lawrence Erlbaum Associates.

Rescorla, R.A. (2003). Elemental and Configural Encoding of the Conditioned Stimulus. *Quarterly Journal of Experimental Psychology, 56B(2),* 161-176.

Rescorla, R.A., & Wagner, A.R. (1972). A theory of Pavlovian conditioning: variations in the effectiveness of reinforcement and nonreinforcement. In Black, A.H. & Prokasy, W.F. (Eds). *Classical conditioning II: Current Theory and Research* (pp. 64-99). New York: Appleton-Century-Crofts.

Reverberi, C., Lavaroni, A., Gigli, G.L., Skrap, M., & Shallice, T. (2005). Specific impairments of rule induction in different frontal lobe subgroups. *Neuropsychologia*, *43*, 460-472.

Rhodes, M.G. (2004). Age-related differences in performance on the Wisconsin Card Sorting Test: a meta-analytic review. *Psychology and Aging*, *19(3)*, 482-494.

Rhodes, M.G., & Kelley, C.M. (2005). Executive processes, memory accuracy, and memory monitoring: an aging and individual difference analysis. *Journal of Memory and Language*, *52(4)*, 578-594.

Ridderinkhof, K.R., Span, M.M., & van der Molen, M.W. (2002). Perseverative behavior and adaptive control in older adults: Performance monitoring, rule induction, and set shifting. *Brain & Cognition*, *49(3)*, 382-401.

Riegal, K.F., & Riegal, R.M. (1972). Development, drop, and death. *Developmental Psychology*, *6*, 306-319.

Riege, W.H. (1971). Environmental influences on brain and behavior of year-old rats. *Developmental Psychobiology*, *4(2)*, 157-167.

Rolls, E.T. (1999). The representation of space in the primate hippocampus, and its role in memory. In N. Burgess, & K.J. Jeffery, *Hippocampal and Parietal Foundations of Spatial Cognition* (pp. 320-344). London: Oxford University Press, 1999.

Rolls, E.T. (2000). Memory systems in the brain. *Annual Review of Psychology*, *51*, 599-630.

Rolls, E.T. (2004). The functions of the orbitofrontal cortex. *Brain & Cognition*, *55(1)*, 11-29.

Ronnlund, M., Nyberg, L., Backman, L., & Nilsson, L.G. (2005). Stability, growth and decline of adult life span development of declarative memory: cross-sectional and longitudinal data from a population based study. *Psychology and Aging*, *20(1)*, 3-18.

Rosen, A.C., Prull, M.W., O'Hara, R., Race, E.A., Desmond, J.E., Glover, G.H., Yesavag, J.A., & Gabrieli, J.D.E. (2002). Variable effects of aging on frontal lobe contributions to memory. *Neuroreport: For Rapid Communication of Neuroscience Research*, *13(18)*, 2425-2428.

Rosnick, C.B., Small, B.J., Graves, A.B., & Mortimer, J.A. (2004). The association between health and cognitive performance in a population based study of older adults: the Charlotte County Healthy Aging Study (CCHAS). *Aging Neuropsychology & Cognition*, *11(1)*, 89-99.

Ross, C.E., & Mirowsky, J. (1989). Explaining the social patterns of depression: Control and problem solving: or Support and talking? *Journal of Health and Social Behavior*, *30(2)*, 206-219.

Rudy, J.W., & Sutherland, R.J. (1989). The hippocampal formation is necessary for rats to learn and remember configural discriminations. *Behavioral Brain Research*, *34(1-2)*, 97-109.

Rudy, J.W. & Sutherland, R.J. (1995). Configural association theory and the hippocampal formation: an appraisal and reconfiguration. *Hippocampus*, *5*, 375-389.

Rumelhart, D.E., Hinton, G.E., & Williams, R.J. (1986). Learning internal representations by error backpropogation. In Rumelhart, D.E., McClelland, J.L. (Eds.), *Parallel Distributed Processing: Adventures in the Microstructure of Cognition* (pp. 318-362). Cambridge, MA: MIT Press.

Rympa, B., Prabhakaran, V., Desmond, J.E., & Gabrieli, J.D.E. (2001). Age differences in prefrontal cortical activity in working memory. *Psychology and Aging*, *16(3)*, 971-384.

Saavedra, M.A. (1975). Pavlovian conditioning in the rabbit. *Learning and Motivation*, *6(3)*, 314-326.

Sachs, L.B. (1969). Effects of a stimulus comparison during discrimination training on subsequent transposition and generalization gradients. *Psychonomic Science*, *14*, 128-135.

Salat, D.H., Kaye, J.A., & Janowsky, J.S. (2002). Greater orbital prefrontal volume selectively predicts worse working memory performance in older adults. *Cerebral Cortex*, *12(5)*, 494-505.

Salthouse, T.A. (1991). *Theoretical Perspectives on Cognitive Aging*. Hillsdale, NJ: Lawrence Erlbaum.

Salthouse, T.A. (1992). Why do adult age differences increase with task complexity? *Developmental Psychology*, *28 (5)*, 905-918.

Salthouse, T.A. (1993). Speed mediation of adult age differences in cognition. *Developmental Psychology*, *29*, 722-738.

Salthouse, T.A. (1994). Aging associations: Influence of speed in associative learning. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *20*, 1486-1503.

Salthouse, T.A. (1996). The processing speed theory of adult age differences in cognition. *Psychological Review*, *103 (3)*, 403-428.

Salthouse, T.A. (2000). Steps towards the explanation of adult age differences in cognition. In Perfect, T.J., & Maylor, E.A. (Eds.), *Models of Cognitive Aging* (pp. 19-49). Oxford: Oxford University Press.

Salthouse, T.A. (2001). Structural models of the relations between age and measures of cognitive functioning. *Intelligence*, *29*, 93-115.

Salthouse, T.A. & Babcock, R.L. (1991). Decomposing adult age differences in working memory. *Developmental Psychology*, *27*, 763-776.

Salthouse, T.A., & Coon, V.E. (1993). Influence of task-specific processing speed on age differences in memory. *Journals of Gerontology: Psychological Sciences*, *48B*, 245-255.

Salthouse, T.A. & Meinz, E.J. (1995). Aging, inhibition, working memory, and speed. *Journals of Gerontology: Psychological Sciences*, *50B*, 297-306.

Salthouse, T.A., Mitchell, D.R., Skovronek, E., & Babcock, R.L. (1989). Effects of adult age and working memory on reasoning and spatial ability. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *15*, 507-516.

Salvatierra, A.T., & Berry, S.D. (1989). Scopolamine disruption of septo-hippocampal activity and classical conditioning. *Neuroscience*, *103(4)*, 715-721.

Schaie, K.W. (1979). The primary mental abilities in adulthood: an exploration into the development of psychometric intelligence. In Baltes, P.B. & Brim, O.G. (Eds.), *Lifespan Development and Behaviour, Volume 2* (pp. 67-115). New York: Prentice-Hall.

Schaie, K.W. (1983). The Seattle longitudinal study: a 21 year exploration of psychometric intelligence in adults. In Schaie, K.W. (Ed.) *Longitudinal Studies of Adult Psychological Development* (pp. 64-135). New York: Guilford Press.

Schaie, K.W. (1985). *Manual for the Schaie-Thurstone Adult Mental Abilities Test (STAMAT)*. Palo Alto, CA: Consulting Psychologists Press.

Schaie, K.W. (1986). Beyond calendar definitions of age, time, and cohort: the general developmental model revisited. *Developmental Review*, *6*, 252-277.

Schaie, K.W. (1994). The course of adult intellectual development. *American Psychologist*, *49(4)*, 304-313.

Schaie, K.W., & Hertzog, C. (1983). Fourteen year cohort-sequential analyses of adult intellectual development. *Developmental Psychology*, *19*, 531-543.

Schaie, K.W., & Hertzog, C. (1986). Toward a comprehensive model of adult intellectual development: Contributions of the Seattle Longitudinal Study. In Sternberg, R. (Ed.), *Advances in Human Intelligence* (pp. 79-118). Hillsdale, NJ: Lawrence Erlbaum Associates.

Schuff, N., Amend, D.L., Knowlton, R., Norman, D., Fein, G., & Weiner, M.W. (1999). Age related metabolite changes and volume loss in the hippocampus by magnetic resonance spectroscopy and imaging. *Neurobiology of Aging*, *20*, 279-285.

Scoville, W.B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery, & Psychiatry*, *20*, 11-21.

Shallice, T., & Burgess, P. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain*, *114*, 727-741.

Shan, Z.Y., Liu, J.Z., Sahgal, V., Wang, B., & Yue, G.H. (2005). Selective atrophy of left hemisphere and frontal lobe of the brain in old men. *Journals of Gerontology: Series A: Biological Sciences & Medical Sciences*, *60(2)*, 165-174.

Shanks, D.R., Charles, D., Darby, R.J., & Azmi, A. (1998). Configural processes in human associative learning. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *24 (6)*, 1353-1378.

Shanks, D.R., & Darby, R.J. (1998). Feature and rule based generalization in human associative learning. *Journal of Experimental Psychology: Animal Behaviour Processes*, *24 (4)*, 405-415.

Shanks, D.R., Darby, R.J., & Charles, D. (1998). Resistance to interference in human associative learning: evidence of configural processing. *Journal of Experimental Psychology: Animal Behaviour Processes*, *24 (2)*, 136-150.

Shimamura, A.P., Berry, J.M., Mangels, J.A., Rusting, C.L., & Jurica, P.L. (1995). Memory and cognitive abilities in university professors: Evidence for successful aging. *Psychological Science*, *6(5)*, 271-277.

Siedlecki, K.L., Salthouse, T.A., & Berish, D.E. (2005). Is there anything special about the aging of source memory? *Psychology and Aging*, *20(1)*, 19-32.

Simons, J.S., Dodson, C.S., Bell, D., & Schacter, D.L. (2004). Specific- and partial-source memory: Effects of aging. *Psychology & Aging*, *19(4)*, 689-694.

Skinner, B.F. (1938). *The Behavior of Organisms*. New York: Appleton-Century-Crofts.

Small, B.J., Rosnick, C.B., Fratiglioni, L., & Backman, L. (2004). Apolipoprotein E and cognitive performance: a meta-analysis. *Psychology and Aging*, *19(4)*, 592-600.

Soloman, P., & Gottfried, K. (1981). The septohippocampal cholinergic system and classical conditioning of the rabbit's nictitating membrane response. *Journal of Comparative and Physiological Psychology*, *95(2)*, 322-330.

Soloman, P., Groccia-Ellison, M., Flynn, D., Mirak, J., Edwards, K., Dunehew, A., & Stanton, M. (1993). Disruption of human eyeblink conditioning after central cholinergic blockade with scopolamine. *Behavioral Neuroscience*, *107(2)*, 271-279.

Soloman, P., Soloman, S., Van der Schaaf, E., & Perry, H. (1983). Altered activity in the hippocampus is more detrimental to classical conditioning than removing the structure. *Science*, *220I,* 329-331.

Spearman, C. (1927). *The Abilities of Man*. London: MacMillan.

Spence, K.W. (1936). The nature of discrimination learning in animals. *Psychological Review*, *43*, 427-449.

Spence, K.W. (1937). The differential response in animals to stimuli varying in a single dimension. *Psychological Review*, *44*, 430-444.

Spencer, W.D., & Raz, N. (1995). Differential effects of aging on memory for content and context: a meta-analysis. *Psychology and Aging, 10 (4)*, 527-539.

Squire, L.R., & Zola-Morgan, S. (1983). The neurology of memory: the case for correspondence between the findings for human and non-human primate. In Deutsch, J.A. (Ed.), *The Physiological Basis of Memory, 2nd Edition* (pp. 199-268). New York: Academic Press.

Squire, L.R., & Zola-Morgan, S. (1985). Neuropsychology of memory: New links between humans and experimental animals. In Olton D., Corkin, S., & Gamsu, E. (Eds.), *Conference on Memory Dysfunctions* (pp. 137-149). New York: New York Academy of Sciences.

Squire, L.R. (1992). Memory and the hippocampus: A synthesis of findings with rats, monkeys, and humans. *Psychological Review*, *99*, 195-231

Starr, J.M., McGurn, B., Whiteman, M., Pattie, A., Whalley, L.J., & Deary, I.J. (2004). Life long changes in cognitive ability are associated with prescribed medications in old age. *International Journal of Geriatric Psychiatry*, *19(4)*, 327-332.

Storandt, M. (1976). Speed and coding effects in relation to age and ability level. *Developmental Psychology*, *12*, 177-178.

Storandt, M. (1977). Age, ability level and scoring the WAIS. *Journal of Gerontology*, *32*, 175-178.

Streisand, R.M., Rodrigue, J.R., & Sears, S.F. (1999). A psychometric normative database for pre-liver transplantation evaluations: the Florida cohort 1991-1996. *Psychosomatics*, *40 (6)*, 479-485.

Stroop, J.R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, *18*, 643-662.

Stuart-Hamilton, I. (1994). *The Psychology of Ageing: An Introduction.* London: Jessica Kingsley.

Stuart-Hamilton, I., & McDonald, L. (1998). Aging and the Bridges of Konigsberg problem: no age changes in perseverance. *Educational Gerontology*, *24*, 225-232.

Stuart-Hamilton, I., & McDonald, L. (2001). Do we need intelligence? Some reflections on the perceived importance of “g”. *Educational Gerontology*, *27*, 399-407.

Stuart-Hamilton, I., Nayak, L., & Priest, L. (2006). Intelligence, belief in the paranormal, knowledge of probability and aging. *Educational Gerontology*, *32*, 173-184.

Suret, M.B., & McLaren, I.P.L. (2003). Representation and discrimination along an artificial dimension. *Quarterly Journal of Experimental Psychology, 56B,* 30-42.

Sutherland, R.J., & Rudy, J.W. (1989). Configural association theory: The role of the hippocampal formation in learning, memory, and amnesia. *Psychobiology*, *17(2)*, 129-144.

Terry, A.V., & Buccafusco, J.J. (2003). The cholinergic hypothesis of age and Alzheimer's disease-related cognitive deficits: Recent challenges and their implications for novel drug development. *The Journal of Pharmacology and Experimental Therapeutics*, *306(3)*, 821-827.

Thorndike, E.L., (1911). *Animal Intelligence*. NewYork: MacMillan.

Thorpe, S. J., Rolls, E. T., & Maddison, S. (1983). The orbitofrontal cortex: Neuronal activity in the behaving monkey. *Experimental Brain Research*, *49*, 93–115.

Thurstone, T.G. (1958). *Manual for the SRA Primary Mental Abilities*. Chicago: Science Research Associates.

Treves, A., & Rolls, E. (1992). Computational constraints suggest the need for two distinct input systems to the hippocampal CA3 network. *Hippocampus*, *2(2)*, 189-200.

Tulving, E. (1983). *Elements of Episodic Memory*. Oxford: Clarendon.

Tulving, E. (2002). Episodic memory: From mind to brain. *Annual Review of Psychology*, *53(1)*, 1-25.

Turner, A.M, & Greenough, W.T. (1985). Differential rearing effects on rat visual cortex synapses: I. Synaptic and neuronal density and synapses per neuron. *Brain Research*, *329*, 195-203.

Valentijn, S.A.M, van Boxtel, M.P.J, van Hooren, S.A.H., Bosma, H., Beckers, M.D., Ponds, R.W.H.M., & Jolles, J. (2005). Change in sensory functioning predicts change in cognitive functioning: Results from a 6-year follow-up of the Maastricht Aging Study. *Journal of the American Geriatric Society*, *53*, 374-380.

van Boxtel, M.P.J, Buntinx, F., Houx, P.J., Metsemakers, J.F.M., Knottnerus, A. & Jolles, J. (1998). The relationship between morbidity and cognitive performance in a normal aging population. *Journals of Gerontology: Series A: Biological Sciences and Medical Sciences*, *53A (2)*, 147-154.

van Hooren, S.A.H., Valentijn, S.A.M., Bosma, H., Ponds, R.W.H.M., van Boxtel, M.P.J., & Jolles, J. (2005). Relation between health status and cognitive functioning: A 6-year follow-up of the Maastricht Aging Study. *Journals of Gerontology: Series B: Psychological Sciences & Social Sciences*, *60(1)*, 57-60.

Vanderwolf, C. H., & Leung, L-W. S. (1983). Hippocampal rhythmical slow activity: a brief history and the effects of entorhinal lesions and phencyclidine. In W. Seifert (Ed.), *Neurobiology of the Hippocampus* (pp. 225–302). Academic Press: London.

Verhaeghen, P., & Basak, C. (2005). Ageing and switching of the focus of attention in working memory: Results from a modified N-Back task. *Quarterly Journal of Experimental Psychology, 58(A)*, 134-154.

Verhaeghen, P., & Cerella, J. (2002). Aging, executive control, and attention: a review of meta-analyses. *Neuroscience and Biobehavioral Reviews, 26*, 849-857.

Volkow, N.D., Gur, R.C., Wang, G-J., Fowler, J.S., Moburg, P.J., Ping, Y.S., Hitzemann, R., Smith, G., & Logan, J. (1998). Association between decline in brain dopamine activity with age and cognitive and motor impairment in healthy individuals. *American Journal of Psychiatry*, *155(3)*, 344-349.

Wagner, A.R. (1969). Incidental stimuli and discrimination learning. In Gibert, R.M. and Sutherland, N.S. (Eds.), *Animal Discrimination Learning* (pp. 83-111). London: Academic Press.

Wagner, A.R. (2003). Context sensitive elemental theory. *Quarterly Journal of Experimental Psychology, 56B*, 7-29.

Walhovd, K.B., Fjell, A.M., Reinvang, I., Lundervold, A., Fischl, B., Salat, D., Quinn, B.T., Makris, N., & Dale, A.M. (2005). Cortical volume and speed of processing are complementary in prediction of performance intelligence. *Neuropsychologia*, *43*, 704-713.

Wasserman, E.A. (1990). Attribution of causality to common and distinctive elements of compound stimuli. *Psychological Science*, *1*, 298-302.

Wasserman, E.A., & Miller R.R. (1997). What's elementary about associative learning? *Annual Review of Psychology*, *48*, 573-607.

Waters, G., & Caplan, D. (2005). The relationship between age, processing speed, working memory capacity and language comprehension. *Memory*, *13(3/4)*, 403-413.

Watson, J.B. (1930). *Behaviorism*. New York: Norton.

Wearden, J.H. (1991). Human performance on an analogue of an interval bisection task. *Quarterly Journal of Experimental Psychology*, *43b*, 59-81.

Wearden, J.H. (1992). Temporal generalization in humans. *Journal of Experimental Psychology: Animal Behavior Processes*, *21*, 318-330.

Wearden, J.H, Wearden, A.J., & Rabbitt, P.M.A. (1997). Age and IQ effects on stimulus and response timing. *Journal of Experimental Psychology: Human Perception and Performance*, *23(4)*, 962-979.

Wechsler, D. (1955). *Manual for the Wechsler Adult Intelligence Scale.* New York, The Psychological Corporation.

Wechsler, D. (1958). *Measurement of Adult Intelligence*. Baltimore, MD: Williams and Wilkins.

Wechsler, D. (1981). *Manual for the Wechsler Adult Intelligence Scale – Revised*. New York: The Psychological Corporation.

Wechsler, D. (1987). *Manual for Wechsler Memory Scale – Revised*. New York: The Psychological Corporation.

Wechsler, D. (1997). *Weschsler Adult Intelligence Scale-III*. San Antonio, TX: The Psychological Corporation.

Weisberg, R.W. (1996). Prolegomena to theories of insight in problem solving: A taxonomy of problems. In Sternberg, R.J., & Davidson, J.E. (Eds.), *The Nature of Insight* (pps. 157-196). Cambridge, MA: MIT Press.

Weisberg, R.W., & Alba, J.W. (1981). An examination of the alleged role of fixation in the solution of several insight problems. *Journal of Experimental Psychology: General, 110(2),* 169-192.

Welford, A.T. (1958). *Ageing and Human Skill*. London: Oxford University Press.

West, M.J. (1993). Regionally specific loss of neurons in the aging human hippocampus. *Neurobiology of Aging*, *14*, 287-293.

West, R., & Craik, F.I.M. (2001). Influences on the efficiency of prospective memory in younger and older adults. *Psychology & Aging*, *16(4)*, 682-696

West, R.L. (1996). An application of prefrontal cortex function theory to cognitive aging. *Psychological Bulletin*, *120 (2)*, 272-292.

White, N., & Cunningham, W.R. (1988). Is terminal drop pervasive or specific? *Journal of Gerontology*, *43(6)*, 141-144.

Widrow, B., & Hoff, M. (1960). Adaptive switching circuits. *Institute of Radio Engineers, Western Electronic Show and Convention Record*, *4*, 96-104

Willis, S.L., & Schaie, K.W. (1986). Training the elderly on the ability factors of spatial orientation and inductive reasoning. *Psychology and Aging*, *1*, 239-247.

Wilson, B.A., Alderman, N., Burgess, P., Emslie, H., & Evans, J.J. (1996). *Behavioural Assessment of the Dysexecutive Syndrome*. Bury St. Edmunds: Thames Valley Testing Company.

Wilson, P.M., Boumphrey, P., & Pearce, J.M. (1992). Restoration of the orienting response to a light by a change in its predictive accuracy. *Quarterly Journal of Experimental Psychology*, *44B*, 17-36.

Wingfield, A., Poon, L.W., Lombardi, L., & Lowe, D. (1985). Speed of processing in normal aging: Effects of speech rate, linguistic structure, and processing time. *Journal of Gerontology*, *40*, 579-585.

Woodruff-Pak, D.S. (1997). *The Neuropsychology of Aging*. Malden, MA: Blackwell.

Yerkes, R.M. (1921). Psychological examining in the United States army. *Memoirs of the National Academy of Sciences*, *15*, 1-877.

Young, M.E., Wasserman, E.A., Johnson, J.L., & Jones, F.L. (2000). Positive and negative patterning in human causal learning. *Quarterly Journal of Experimental Psychology*, *53B(2)*, 121-138.

Zacks, R.T., & Hasher, L. (1994). Directed ignoring: Inhibitory regulation of working memory. In D. Dagenbach, & T.H. Carr (Eds.), *Inhibitory Processes in Attention, Memory, and Language* (pp. 241-264). San Diego, CA: Academic Press.

Zacks, R.T., Hasher, L., & Li, K.Z.H. (2000). Human Memory. In Craik, F.I.M. & Salthouse, T.A. (Eds.), *The Handbook of Aging and Cognition* (pp. 293-357). Mahwah, NJ: Erlbaum.

Zelinski, E.M., Crimmins, E., Reynolds, S., & Seeman, T. (1998). Do medical conditions affect cognition in older adults? *Health Psychology*, *17 (6)*, 504-512.

Zimprich, D. (2002). Cross sectionally and longitudinally balanced effects of processing speed on intellectual abilities. *Experimental Aging Research*, *28*, 231-251.

Zola-Morgan, S., Squire, L.R., & Amaral, D.G. (1986). Human amnesia and the medial temporal region: Enduring memory impairment following a bilateral lesion limited to field CA 1 of the hippocampus. *The Journal of Neuroscience*, *6 (10)*, 2950-2967.